ARCHIVES OF SURGERY

VOLUME 57 OCTOBER 1948 NUMBER 4

COPYRIGHT, 1949, BY THE AMERICAN MEDICAL ASSOCIATION

TREATMENT OF THYROIDITIS

GEORGE CRILE Jr., M.D.
CLEVELAND

THE THREE main and distinct clinical types of thyroiditis are (1) the subacute (pseudotuberculous or giant cell) variety, (2) struma lymphomatosa (Hashimoto's thyroiditis) and (3) Riedel's struma.[1] Subacute thyroiditis resolves promptly and completely in response to roentgen therapy; some, and possibly most, cases of struma lymphomatosa respond favorably to roentgen rays, but Riedel's struma is unaffected by irradiation.

SUBACUTE THYROIDITIS

Subacute thyroiditis is a self-limited disease of unknown causation. It has a variable course of weeks or months and eventually subsides without treatment and without significant interference with the function of the thyroid.

This type of thyroiditis has been variously named tuberculous, pseudotuberculous or giant cell thyroiditis because of the histologic appearance of pseudotubercles with giant cells. Tubercle bacilli cannot be demonstrated in the lesions, and the cause of the disease is unknown. Bacteria have not been demonstrated in the thyroid. The possibility that it represents a virus infection has not been excluded. The pseudotubercle or giant cell reaction represents a reaction of wandering cells to colloid, which they appear to be phagocytosing. The sedimentation rate is consistently elevated.

Many surgeons do not operate on patients with subacute thyroiditis and hence are not aware that this well recognized clinical entity is, from the histologic standpoint, identical with pseudotuberculous or giant cell thyroiditis. In order to prove to my own satisfaction that the two diseases are the same, I have analyzed 15 cases of subacute thyroiditis in which roentgen therapy was given and compared the history and physical observations with those in 12 cases in which operation was performed and found that there was no significant difference in the clinical observations in the two groups.[1] To confirm further the fact that the clinical entity of subacute thyroiditis is indeed identical to the pathologic entity

From the Cleveland Clinic, Cleveland.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

1. Crile, G., Jr.: Thyroiditis, Ann. Surg. **127**:640-654 (April) 1948.

of giant cell or pseudotuberculous thyroiditis, biopsy specimens of the thyroid were taken in 2 typical cases of subacute thyroiditis, and the patients were then treated with roentgen rays. The biopsy specimens showed typical giant cell or pseudotuberculous thyroiditis; the response to roentgen therapy was prompt, complete and typical of that of subacute thyroiditis.

TREATMENT

Subacute thyroiditis responds promptly and completely to roentgen therapy. Six hundred to 800 r usually suffices to effect resolution in a few weeks. The pain and tenderness subside in a few days. The average time at which 15 patients treated with roentgen rays were considered to be entirely well was nineteen days after the start of treatment. By this time the thyroid is rarely either tender or palpably enlarged. In 2 of our cases, two or more courses of treatment over a period of three months were required before a complete cure was effected. In 3 cases the patients considered themselves well in one week.

Thyroidectomy is a satisfactory means of controlling subacute thyroiditis, but since the disease is essentially self limited and since roentgen rays effect such prompt and complete resolution, operation is not often indicated.

REPORT OF CASES

CASE 1.—The patient (previously reported [1]) was a woman, 32 years old, who had a hard tender painful enlargement of the thyroid gland, of two months' duration. Biopsy showed chronic thyroiditis, with decided granulomatous reaction to colloid (so-called pseudotuberculous thyroiditis). Foreign body giant cells were present.

A diagnosis of subacute thyroiditis was made, and roetgen therapy was started. Seven hundred roentgen units were given to the region of the thyroid in five treatments. Nine days after the first treatment the patient stated that he felt entirely well. There was no pain or tenderness of the thyroid. Although the gland had returned to normal size the right lobe was still fairly firm. Four weeks later, palpation of the thyroid revealed no abnormalities and the patient remained well.

CASE 2.—The patient was a woman, 35 years of age, who complained of a painful tender lump in the left lobe of the thyroid, which had been present for three weeks. The pulse rate was 150 and the basal metabolic rate + 18 per cent. The thyroid was enlarged to twice its normal size and was hard.

A diagnosis of subacute thyroiditis was made, and 700 r was given over the thyroid. A month after the last treatment the right lobe of the thyroid was normal to palpation and the left lobe was a little smaller but was still hard. Biopsy specimens were taken of both lobes of the thyroid and showed the typical picture of subacute thyroiditis with focal granulomatous reaction to colloid, slight to decided fibrosis and moderate lymphoid hyperplasia (so-called pseudotuberculous thyroiditis).

A month after the biopsy and two months after roentgen therapy was started, the thyroid was no longer palpable and the patient felt well.

COMMENT

The clinical features of these cases and the response of the thyroid to roentgen therapy are typical of those encountered in other cases of

subacute thyroiditis. Biopsy of the thyroid showed changes characteristic of the so-called pseudotuberculous or giant cell thyroiditis. There can be little doubt that the clinical disease, subacute thyroiditis, is from the pathologic point of view pseudotuberculous or giant cell thyroiditis. This disease responds promptly to roentgen therapy and does not necessitate thyroidectomy.

STRUMA LYMPHOMATOSA

Struma lymphomatosa is a progressive disease of the thyroid, possibly associated with systemic disorders.[2] There is extensive acidophilic degeneration of the epithelial elements of the thyroid and replacement by lymphoid and fibrous tissue. Hypothyroidism, or at least a peculiar type of hypometabolism that does not always respond specifically to treatment with thyroid, is likely to develop. The cause of the disease is unknown. It does not progress to Riedel's struma,[3] nor is it the end result of subacute thyroiditis. An excellent description of this disease was given by Joll in 1939.[4]

Until recently the diagnosis of struma lymphomatosa was rarely established prior to operation, and hence there was little opportunity to study its response to irradiation. Schilling [5] reported a case of struma lymphomatosa that responded to roentgen therapy.

In the last 3 cases that I have seen the diagnosis of struma lymphomatosa was established before operation, and in the last 2 cases I have performed only a biopsy and have treated the patient with roentgen rays. In the first case there was no immediate effect on the thyroid, but in six months the gland was not palpable. In the second there was prompt and complete resolution of the enlarged thyroid.

REPORT OF CASES

CASE 1.—The patient, a woman, 44 years of age, entered the clinic with complaints referable to nearly every system of the body. She had been receiving medical care constantly for fifteen years. For four years prior to this examination she had noted an intermittent tender swelling of the neck, with pain radiating up to the ears. Among her other complaints were headache, fatigue, gain of 50 pounds (about 23 Kg.) of weight, swelling of the entire body, tightness of the shoulders, numbness of the extremities, dizziness, nervousness and shortness of breath.

---

2. Graham, A.: Struma Lymphomatosa (Hashimoto), Tr. Am. A. Study Goiter, 1940, pp. 22-251.

3. Graham, A., and McCullagh, E. P.: Atrophy and Fibrosis Associated with Lymphoid Tissue in Thyroid; Struma Lymphomatosa (Hashimoto), Arch. Surg. **22**:548-567 (April) 1931.

4. Joll, C. A.: Pathology, Diagnosis, and Treatment of Hashimoto's Disease (Struma Lymphomatosa), Brit. J. Surg. **27**:351-389 (Oct.) 1939.

5. Schilling, J. A.: Struma Lymphomatosa, Struma Fibrosa, and Thyroiditis, Surg., Gynec. & Obst. **81**:533-550 (Nov.) 1945.

Results of examination were essentially negative except for a blood pressure of 148 systolic and 112 diastolic, obesity and a hard enlargement of the entire thyroid to about twice its normal size. The left lobe was larger than the right. The gland was only slightly tender. She was a little hoarse.

Examination of the larynx showed thickening of the anterior end of the right vocal cord, consistent with chronic laryngitis. Results of urinalysis, blood counts, the level of blood sugar, Wassermann and Kahn tests and a roentgenogram of the chest were within normal limits. The basal metabolic rate was —22 per cent, and the level of blood cholesterol was 120 and on a later test 179 mg. per cubic centimeter.

A diagnosis of struma lymphomatosa was made and confirmed by biopsy. The specimen showed dense fibrous tissue with several broad patches of lymphocytes and plasma cells intermingled with a few epithelial elements resembling thyroid epithelium. Other fragments showed similar architecture with broad patches of dense lymphocytic infiltration, one area including some patches of acidophilic epithelium.

Roentgen therapy was advised, and 700 r was given to each lobe of the thyroid. The symptoms of pressure were relieved promptly, but, two months after treatment was started, there was little diminution in the size of the thyroid and it was still firm.

Six months after the beginning of treatment, the thyroid was not palpable and the patient remained free of local symptoms. The systemic symptoms persisted and did not respond to treatment with thyroid and amphetamine.

CASE 2.—The patient was a woman, 53 years of age, who complained of a "tight choking bursting" sensation in the throat, dating back to an injury sustained three months before entering the clinic. She had also noted lack of energy, drowsiness, sensitivity to cold, thickness of speech, constipation and flatulence.

Examination showed typical myxedema and a firm diffuse enlargement of the entire thyroid. The basal metabolic rate was —31 per cent, and the cholesterol level 316. A clinical diagnosis of struma lymphomatosa with associated myxedema was made and confirmed by biopsy. The pathologist reported observing rare thyroid follicles which were small, and had epithelial cells which were larger than usual with eosinophilic-staining characteristics. Little recognizable colloid remained. There was decided infiltration by lymphocytes and plasma cells, with occasional formation of lymph follicles. The stroma consisted largely of dense collagenous fibers.

A total of 1,550 r was given to the thyroid in eight treatments. The choking sensations subsided promptly and eight days after the beginning of treatment the gland was much softer and was considerably reduced in size. A month later, it was barely palpable and the patient, who was taking thyroid, felt much improved and had no symptoms other than nervousness and fatigue.

COMMENT

Neither roentgen therapy nor thyroidectomy have any effect on the systemic symptoms associated with struma lymphomatosa. The tendency to hypometabolism and its associated symptoms of weakness and exhaustion are not specifically corrected by feeding thyroid. Struma lymphomatosa appears to be a systemic disease. Roentgen therapy of the thyroid and thyroidectomy are nothing more than symptomatic treatment, which alleviates the local symptoms arising from the goiter. Thyroidec-

tomy does no more to alleviate the systemic symptoms than does roentgen therapy, and if the local symptoms can be controlled by irradiation there is no need to remove the goiter. . How frequently struma lymphomatosa will respond to irradiation has not been established.

RIEDEL'S STRUMA

Riedel's struma is a chronic proliferating fibrosing inflammatory process, involving usually one lobe, but sometimes both lobes, of the thyroid and extending to involve the trachea and the muscles, fascia, nerves and vessels in the vicinity of the thyroid. It produces a bulky tumor that may be indistinguishable preoperatively from an inoperable carcinoma. It is not the end result of either subacute thyroiditis or struma lymphomatosa, but is a separate entity the cause of which is unknown. No specific organisms have been isolated from this lesion. In many instances the inflammatory reaction appears to center about a degenerating adenoma, and this may be a clue to its cause. Riedel's struma affects women more often than men and tends to occur beyond the age of 50.

TREATMENT

An adequate trial of roentgen therapy was made in 3 of the cases of Riedel's struma, without significant results. In 2 cases in which one lobe had been removed and a recurrence later took place on the other side, the roentgen therapy seemed to prevent further proliferations, although there was no change in the size of the tumor. In a third case, in spite of treatment with 2,550 r, pain and symptoms of compression continued, hypothyroidism developed and the process extended to involve the parathyroids and produced tetany. One must assume, therefore, that roentgen rays have little to offer in the treatment of this disease.

Complete surgical removal of the involved portion of the thyroid may be rendered utterly impossible by the extent of the extracapsular fibrosis. Serious damage to the trachea, carotid sheath or recurrent nerves may take place if the true nature of the lesion is not recognized and radical extirpation is attempted. In this disease one must often be content to do the best one can, within the bounds of safety, to relieve obstruction.

On the other hand, if one remembers the fact that at the center of the fibrosed lobe there is often a degenerating adenoma and that around this adenoma the fibrous tissue is deposited in concentric laminations which afford natural cleavage planes, it is often possible, without jeopardizing the vital structures adherent to the capsule of the thyroid, to split the lobe open and enucleate this central core. The results following this simple procedure have been excellent in the 3 cases in which I have found it practicable. Pressure symptoms have been relieved; the bulk of the tumor has been strikingly diminished, and the progress of the inflam-

matory and productive process appears to be arrested. It is well to remember that the severest obstruction to respiration usually is associated with retrotracheal adenomas that compress the trachea from behind and that this can be demonstrated before operation by a lateral roentgenogram of the trachea. If the surgeon is not aware of the retrotracheal tumor he is likely to overlook it in a thyroid which cannot be mobilized and rotated from its bed.

REPORT OF CASES

CASE 1.—The patient (previously reported [1]) was a woman 46 years old. She had noticed an enlargement of the left lobe of the thyroid two years before entry, and a few months later she became hoarse. Three months before entry an attempt was made to do a thyroidectomy, but the surgeon stated that the tumor was an inoperable carcinoma. From a biopsy specimen the lesion was diagnosed as either Riedel's struma or carcinoma.

Symptoms of pressure continued, but there was no loss of weight and no symptoms of systemic disease. Examination showed a stony hard fixed tumor, involving the left lobe of the thyroid and the cervical musculature. The left vocal cord was paralyzed. The basal metabolic rate was + 2 per cent.

A diagnosis of Riedel's struma or inoperable carcinoma was made. At operation it was observed that the left side of the neck was involved in a dense proliferation of fibrous tissue which had invaded the trachea and the prethyroid muscles. The lobe was exposed and the anterior half removed. It was white, brittle and avascular. The cleavage plane surrounding the central adenoma was located, and a degenerating adenoma was shelled out of its capsule of fibrous tissue. The right lobe was normal.

The pathologist reported chronic inflammation and complete replacement of thyroid tissue with fibrous tissue. There was a colloid adenoma in the center of the fibrous mass. Convalescence was uneventful, and the symptoms of pressure were relieved.

Comment.—This case is typical of Riedel's struma, in that (1) the initial diagnosis was carcinoma of the thyroid; (2) there was no systemic reaction or alteration of thyroid function; (3) there was extensive fibrosis of the thyroid and of the perithyroid structures; (4) there was a degenerating adenoma in the center of the fibrous mass, and (5) the symptoms were relieved by enucleation of the adenoma from the surrounding fibrous tissue.

CASE 2.—The patient was a Negro woman 42 years old. Seven years prior to entry a biopsy specimen of the thyroid had shown Riedel's struma. Following this she had been free of symptoms until five weeks before entry, when pain and tenderness developed in the region of the thyroid.

Physical examination showed a hard enlargement of the entire thyroid to four time its normal size. A diagnosis of chronic thyroiditis was made, but no treatment was given..

During the next four years the patient experienced a number of episodes of pain and tenderness of the thyroid and was treated with a total of 4,050 r to the thyroid over a period of twenty-two months. Roentgen therapy appeared to relieve the pain, but did not relieve the sensation of pressure or alter the consistency of the thyroid. Finally the sensation of pressure became intolerable, and the patient

became dyspneic from tracheal obstruction. Dr. R. S. Dinsmore removed enough of the thyroid to relieve the compression on the trachea and observed that the muscles, trachea and all the tissues around the thyroid were involved in dense fibrosis consistent with Riedel's struma. The pathologist confirmed the diagnosis of Riedel's struma, having observed that the thyroid was entirely replaced with fibrous tissue showing evidence of chronic inflammation.

Treatment with 2,050 r did not effect any improvement in this patient's symptoms or in the progress of the disease.

CONCLUSIONS

1. Roentgen therapy is the treatment of choice for subacute thyroiditis, and thyroidectomy is rarely, if ever, indicated.

2. When the diagnosis of struma lymphomatosa is made before operation roentgen therapy should be given a trial.

3. If struma lymphomatosa is recognized at the time of operation a very conservative resection of both lobes of the thyroid is recommended.

4. In Riedel's struma, roentgen therapy is of little or no value.

5. In Riedel's struma it is unwise, unnecessary and often dangerous to attempt to remove the entire lobe. If the onion-like concentric laminations in the fibrous tissue surrounding the central degenerating adenoma can be found, these avascular planes can be followed by blunt dissection and the core of the lobe shelled out without disturbing its capsule. Following this procedure, the symptoms are relieved and there is no further proliferation of fibrous tissue.

6. In 2 cases of subacute thyroiditis and 2 cases of struma lymphomatosa proved by biopsy, roentgen therapy was followed by complete resolution of the palpably enlarged thyroid.

7. Three patients with proved Riedel's struma were not benefited by roentgen therapy.

DISCUSSION

Dr. Joseph A. Witter, Detroit: I would like to comment on Dr. Crile's paper "Thyroiditis." During the last two years I have seen 5 cases of the subacute variety of the disease. I do not have a lot to offer, but I would like to state briefly my experience, because it has been at variance with that of Dr. Crile. The lesion in these cases was usually begun unilaterally as a tender, enlarged gland, with considerable systemic malaise, and has progressed slowly. As the original side improved in each case, the other lobe became involved. It is of interest that the second side became involved during the time that the patients were receiving penicillin in large doses and also were taking antithyroid drugs. I feel that perhaps these methods of therapeusis are not too valuable. My patients also received roentgen rays, but treatment was less intensive than recommended by Dr. Crile. The second side became involved during the course of the treatments with roentgen rays.

From this brief experience, I have come to the conclusion that subacute thyroiditis has a clinical course of from four to eight weeks.

SURGICAL MANAGEMENT OF BRANCHIOGENIC CYSTS AND SINUS TRACTS

CHARLES W. McLAUGHLIN Jr., M.D.
OMAHA

LATERAL cervical or branchial cysts and sinus tracts were first described by Hunczowski [1] in 1789. Ascherson [2] in 1832 suggested a relationship between the branchial apparatus and these cysts and sinus tracts on the lateral surface of the neck anterior to the sternomastoid muscle. In 1864 Hensinger [3] introduced the term branchial fistula in describing these lesions.

Much speculation and a great deal of painstaking study have been expended in attempting to clarify the embryologic origin of these branchial lesions. Rabl [4] in the latter part of the nineteenth century deduced that these lateral cervical cysts and sinus tracts were resultant from defects of the second branchial cleft, and this view is today held by many. Wenglowski [5] in 1912 published the results of an exhaustive investigation of this subject covering a period of five years, during which time he studied 246 cadavers and 75 embryos. His observations indicated that the branchial apparatus cannot have remnants below the hyoid bone, and he concluded that branchial fistulas are persistent remnants of the thymic duct originating from the third pharyngeal pouch and pursuing a long oblique downward course in the neck.

Meyer [6] subsequently reviewed Wenglowski's work and accepted his observations. He concluded that the branchial apparatus belongs to the head and not the neck, so that any congenital pathologic remnant must arise along the mandible adjacent to the hyoid bone and nothing below the lower level of the hyoid can have genetic relationship to the branchial apparatus. If one accepts this concept congenital cysts and

---

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

1. Hunczowski, cited by Lahey.[13a]

2. Ascherson, cited by Neel and Pemberton.[14]

3. Hensinger, cited by Neel and Pemberton.[14]

4. Rabl, C.: Zur Bildungsgeschichte des Halses, Prag. med. Wchnschr. **11**:497, 1886.

5. Wenglowski, R.: Ueber die Halsfisteln und Cysten, Arch. f. klin. Chir. **98**:151-208, 1912.

6. Meyer, H. W.: True Branchiogenic Cyst and Fistula of Neck, Arch. Surg. **35**:776-771 (Oct.) 1937.

sinus tracts arising below the hyoid should be classified as lateral cervical cysts or sinus tracts of pharyngothymic duct origin.

These views have been accepted by McNeally [7] and Baumgartner [8] but questioned by Ladd and Gross,[9] Bailey [10] and Sommer and his co-workers.[11] Bailey has reported 1 child and Sommer and his associates 2 soldiers, in each of whom persistent cartilage tethered to a dimple and present since birth was found under the skin along the sternomastoid muscle below the hyoid bone. These observations have been considered to be definite evidence of branchial origin from the second cleft.

Patten [12] in his recent work on embryology stated that these cervical fistulas may arise from either the second or third pouches, in either case the external opening appearing along the medial border of the sternomastoid muscle. He expressed the opinion that fistulas from the second pouch begin internally in the tonsillar fossa and usually make

Branchiogenic Cysts and Sinus Tracts; Series of Cases

Author	Total Cases	Cysts	Sinus	Male	Female	Right	Left	Bilateral
Lahey and Nelson	27	..	..	30%	70%	56%	40%	4%
Bailey	5	..	..	25%	75%	100%	....	...
Neel and Pemberton	319	239	80	54%	46%	54%	44%	2%
Ladd and Gross	69	14	55	....	....	....	....	...
McLaughlin	6	2	4	50%	50%	50%	50%	0%

their exit in the neck near the level of the pharynx. Those from the third pouch leave the pharynx below the level of the hyoid bone and reach the cutaneous surface near the base of the neck.

Since the branchial apparatus appears in the second half of the first month of embryonal life and disappears completely during the second month, it is little wonder that confusion exists in the interpreta-

7. McNeally, R. W.: Cystic Tumors of the Neck, J. Am. Dent. A. **29**:1808-1819, 1942.

8. Baumgartner, C. J., and Stundel, S.: Differential Diagnosis Between Thymic Duct Fistula and Branchial Cleft Fistula, Am. J. Surg. **59**:99-104, 1943.

9. Ladd, W. E., and Gross, R. E.: Congenital Branchiogenic Anomalies, Am. J. Surg. **39**:234-249, 1938.

10. Bailey, H.: The Clinical Aspects of Branchial Fistula, Brit. J. Surg. **21**: 173-183, 1933.

11. Sommer, G. N. J.; Conley, J. J., and Dunlap, H. J.: Cervical Lesions of Branchial Origin, Am. J. Surg. **61**:266-271, 1943.

12. Patten, B. M.: Human Embryology, Philadelphia, The Blakiston Company, 1946, pp. 539-540.

tion of events occurring during this transient period. It must be concluded that for the present the causation is not finally settled.

Branchiogenic cysts and sinuses have been observed more frequently in female than male patients, are slightly commoner on the right side and may occasionally be bilateral (table).

Branchial cysts are probably the commonest lesions to produce a single discrete and movable swelling in the lateral aspect of the neck.[13] While congenital in origin they usually appear in early adult life. Characteristically they present as smooth tense, but mobile, masses in the upper third of the neck anterior to the sternomastoid muscle. Fluctuation is usually demonstrable, but they do not readily transmit light. As they grow they tend to bulge outward instead of inward, in contrast with carotid body tumors. In the absence of infection, the wall of the cyst is relatively thin, lined by stratified epithelium and filled with an opaque or slightly cloudy fluid rich in cholesterol.

Differential diagnosis demands consideration of dermoid cysts, cystic hygromas, lipomas, thyroglossal cysts, tuberculous adenitis, deep cervical abscesses and Hodgkin's disease. Branchiogenic cysts may develop with surprising rapidity, and this fact can add to the confusion in diagnosis.

REPORT OF CASES

CASE 1.—A 20 year old woman was seen in consultation during the seventh month of her first pregnancy, because of a painless cystic mass 5 cm. in diameter in the left upper cervical region which had appeared one month previously. A diagnosis of branchiogenic cyst was made, but excision was deferred until the pregnancy was terminated. She was again seen six weeks later, immediately after delivery. The cystic mass had doubled in size during this period, was very tense and firm, but no signs of inflammation were present. Surgical excision was carried out eight days after delivery without incident (figs. 1 and 2).

With the intervention of infection necessitating aspiration or incision and drainage the cyst wall may become rather thick and adherent to the surrounding structures. Draining sinuses are prone to occur after repeated incision and drainage of these cysts, which may persist until complete surgical excision of the tract and cyst is carried out. Neel and Pemberton [14] have reported that in 80 of their cases of branchiogenic cysts incision and drainage had been carried out one or more times before admission for excision.

---

13. (*a*) Lahey, F. H.: Tumors of the Neck, S. Clin. North America **27**:486-501, 1947. (*b*) Lahey, F. H., and Nelson, H. F.: Branchial Cysts and Sinuses, Ann. Surg. **113**:508-512, 1941. (*c*) Hoover, W. B.: Clinical Conditions Arising from Anomalies or Maldevelopments of the Branchial Arches and Clefts, Ann. Otol. Rhin. & Laryng. **50**:834-850, 1941.

14. Neel, H. B., and Pemberton, J. de J.: Lateral Cervical (Branchial) Cysts and Fistula, Surgery **18**:267-286, 1945.

CASE 2.—A white woman, 32 years of age, was seen, with a history of a tense cystic swelling in the left anterosuperior area of her neck of four months' duration. Under observation elsewhere it had become more swollen and tender. It was incised and a large quantity of brown thick material evacuated. On examination a diagnosis of branchiogenic cyst was made and excision recommended, since the recent incision was healed and no signs of inflammation were present. While the patient was awaiting a suitable date for her hospital admission the cyst again

Fig. 1.—Branchiogenic cyst of two months' duration developing during seventh month of pregnancy.

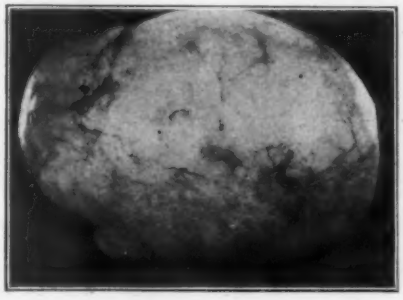

Fig. 2.—Surgical specimen excised from the patient in fig. 1. On cut section it was unilocular and contained milky white fluid.

became acutely inflamed, requiring a second incision and drainage with the liberation of similar material. It again promptly healed, and excision of the entire cyst was carried out without difficulty six weeks later (fig. 3).

This rather large cyst was excised through an incision paralleling the anterior border of the sternomastoid muscle in its upper third. Immediately after operation there was evidence of trauma to the lower branch of the facial nerve. This tem-

porary paresis was transient and disappeared entirely within six weeks. Such a possibility should always be considered in the surgical treatment of lesions in this region because of anatomic variations.

COMMENT

In contrast with branchiogenic cysts the sinus tracts of similar origin are present at birth or become evident very early in life. Often in infancy or childhood they may be present only as a small dimple just

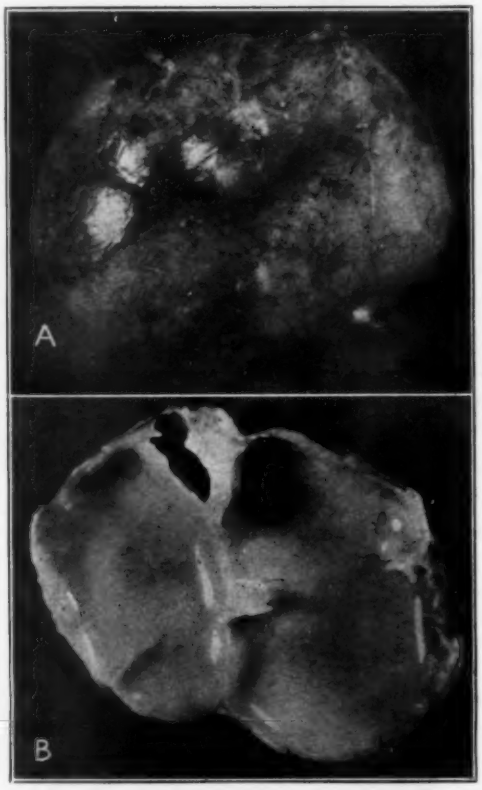

Fig. 3.—*A*, surgical specimen excised in case 2. The somewhat ragged external surface is indicative of the two episodes of acute infection requiring surgical drainage prior to excision. *B*, cut section of the same cyst, showing thickened capsule but a smooth lining. This cyst contained a thick light chocolate-colored material which was sterile on culture.

medial to the sternomastoid above the clavicle. This represents the external opening of the fistulous tract, which may from time to time discharge a few drops of mucous or fetid purulent secretion as secondary infection develops.

Anatomically these fistulous tracts have been divided into three groups: (1) complete fistulas with both an external opening above the

clavicle and lateral to the midline, extending upward and laterally and an internal opening into the pharynx near the lower portion of the tonsillar fossa, (2) incomplete external fistulas which have only an external opening and (3) incomplete internal fistulas which have an opening only into the pharynx.

Complete branchial fistulas are much the commonest form, taking their origin from the pharynx in close proximity to the tonsil and passing out and downward in the neck. As the tract emerges from beneath

Fig. 4.—Injection of complete branchiogenic fistula in the 22 year old man. The tract can be visualized from its external opening above the clavicle to its point of disappearance beneath the mandible.

the digastric muscle it assumes a superficial course in the neck just anterior to the sternomastoid muscle to reach the skin just above the clavicle. In a complete tract the external opening can be observed to move upward in the process of swallowing, and on turning the head to the opposite side the subcutaneous tract can usually be palpated as a firm fibrous cord. These tracts are usually lined with columnar epithelium which may be ciliated in type.

The tract in its downward descent passes very close to the hypoglossal nerve beneath the digastric muscle, and occasionally it will pass between the internal and external carotid arteries, where it will be in close proximity to the vagus nerve. Douglas [15] has recorded a patient able to distend the tract in the neck with air forced from the mouth, but such a phenomenon is rare. A ureteral catheter can frequently be passed up these complete fistulous tracts for several centimeters and injections with iodized poppyseed oil often make visualization of a portion or the tract in its entirety visible on the roentgenogram.

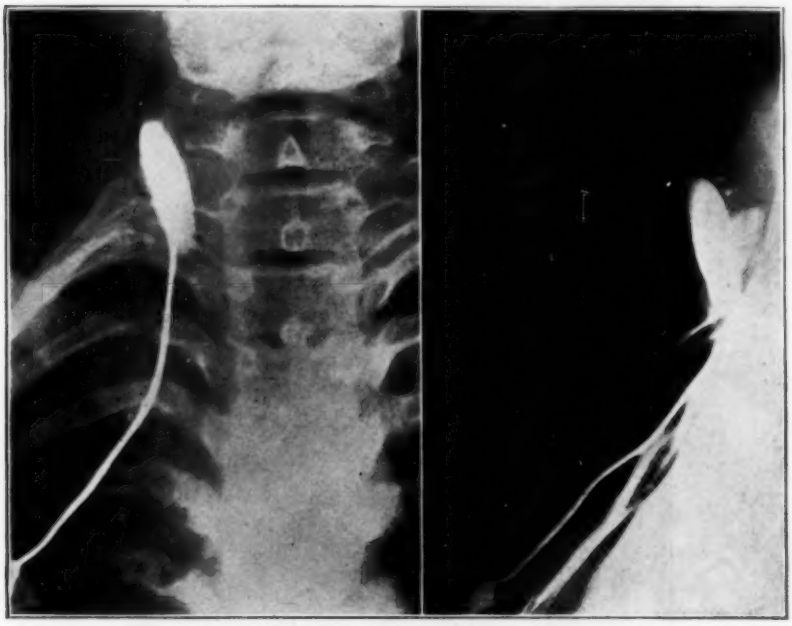

Fig. 5.—Complete branchiogenic fistula in a 19 year old girl with cystic dilatation of lower end of tract. With moderate pressure the injected solution only filled the lower third of the tract which was demonstrated to be complete at operation.

The principal symptoms associated with a complete branchiogenic fistula are a draining sinus, intermittent swelling in the neck on the affected side, dysphagia, drainage in the pharynx with a peculiar taste in the mouth, retraction of the external opening on swallowing and odor from the external drainage.

The injection of sclerosing solutions has been advocated for the obliteration of these fistulous tracts by several observers. This method

15. Douglas, J.: Branchiogenic Cyst with Sinus Leading into Pharynx, Ann. Surg. **67**:240, 1918.

of treatment has not been generally accepted because of the accompanying inflammatory reaction, which may be rather severe, risk of damaging the pharynx with solutions and a persistence of the fibrous tract with a dimpled external opening which remains even after successful obliteration of the tract.

Surgical excision of the fistula is the method of choice, utilizing the stepladder technic originally described by Bailey.[10] Through a small transverse incision incorporating the external opening, the distal tract

Fig. 6.—*A*, branchiogenic sinus tract present since infancy. The scarred external opening is visible together with two secondary draining sites resultant from previous attacks of acute inflammation. *B*, alternative type of incision used in this case to facilitate removal of multiple sinuses.

is dissected upward in the neck as far as possible. A second transverse incision parallel with the first is made at a higher level in the neck. The tract is delivered into the second incision and the dissection continued upward beneath the digastric muscle and medially until the emergence into the pharynx is clearly visualized. In this dissection the hypoglossal nerve should be identified and avoided. The tract is clamped at its junction with the pharynx, doubly ligated and divided.

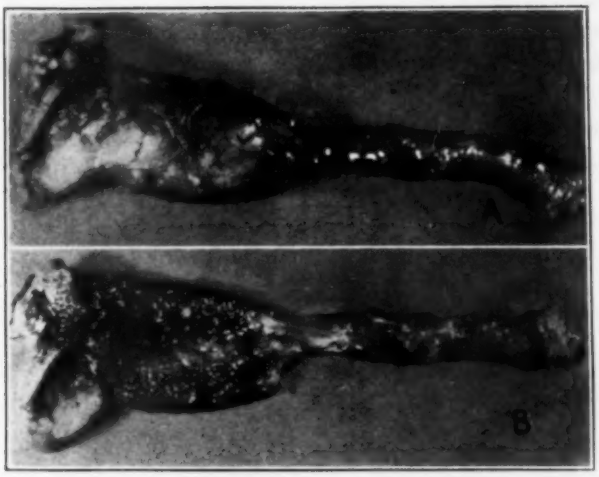

Fig. 7.—A complete branchiogenic fistulous tract after surgical excision. *A*, the tract after excision. *B*, after opening of the tract to demonstrate saccular dilatation of the lower end.

Fig. 8.—Appearance of the incisions three weeks after removal of the complete branchiogenic fistula with the stepladder technic. As a general rule the superior incision can be placed at a higher level than in this case to facilitate the more difficult dissection near the pharynx

The small wounds are closed with a few interrupted sutures, and a drain is left in the upper incision for forty-eight hours. Inversion of the upper opening into the pharynx is not necessary. Excellent cosmetic results without recurrences are to be expected if the entire tract is excised.

I have recently observed four complete branchial fistulas in young adults, three in men and one in a young girl, all but one being on the right side. In each instance the complaints were those of a draining sinus present since early childhood, with troublesome odor from the discharge during recent months. Catheterization of the sinus tracts was attempted in all four cases followed by the injection of iodized poppyseed oil (figs. 4 and 5). Excellent roentgen visualization of the entire tract was obtained in 2 cases, while in the other 2 only the cystic dilatation of the lower third of the fistula could be seen.

All 4 patients were subjected to complete removal of the fistulous tracts with the stepladder type of procedure. The cosmetic results have been very satisfactory and there have been no recurrences and no nerve injuries (figs. 6, 7 and 8).

SUMMARY AND CONCLUSIONS

Branchiogenic cysts and sinus tracts are not infrequently encountered as congenital lesions in the anterior cervical region. The exact embryologic origin of these defects is still not settled, but the principles of surgical therapy are well established. Six additional cases are reported in which the patients underwent surgical treatment without recurrences.

EARLY DIAGNOSIS AND ROENTGEN MANIFESTATIONS OF OBSTRUCTION OF SMALL BOWEL

CLAUDE J. HUNT, M.D.
KANSAS CITY, MO.

THE PURPOSE of this discussion is to emphasize the early clinical manifestations of adhesive obstruction of the small bowel and to call attention to the value of a roentgenogram of the abdomen in the diagnosis, differentiation and localization of the obstruction.

The clinical indications of obstruction of the small bowel are definite and characteristic. They are unlike any other form of abdominal colic and present none of the manifestations of localized disease in either of the four quadrants of the abdomen.

The symptoms and physical signs may be divided into three characteristic manifestations, and they may well be called the "big three" of small bowel obstruction.

They are pain, peristalsis and borborygmus. Although they are often repeated clinical entities and are didactic repetitions, they are worthy of repeated emphasis, because it is only by repetition that fundamental principles of disease become a part of clinical knowledge. Through this process not only do fundamentals become a part of surgeons but reconsideration and discussion open up new fields for thought and speculation and ultimately add to the sum total of knowledge.

Pain in obstruction of the bowel is peculiar unto itself. It is in no way related to any other type of abdominal pain. It is not confined to any quadrant of the abdomen and has little or no localized tenderness or rigidity. It is, therefore, not a localized inflammatory process or localized colic. It is quite the contrary; it is generalized, diffuse and not associated with tenderness or muscle spasm. It is not continuous, but is spasmotic in character. It starts with intestinal cramps, diffuse in character, increasing in intensity and finally culminating in extreme severity, only to subside and recur in a short time. No other form of abdominal colic has this characteristic of diffuseness, periodicity and relatively negative physical observation. Only in strangulated obstruction and adhesive bands attached to the anterior parietal peritoneum will there be localized tenderness or muscle spasm.

From the Surgical Service, Research Clinic and Research Hospital.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

Corresponding with this periodic spasm of pain there occur synchronously visible peristalsis and audible intestinal sounds of particular diagnostic significance.

Peristalsis is sometimes visible in early obstruction and in patients whose abdominal wall is not too thick and obese. It should always be carefully considered, and observations should be made for its presence during the time of the intestinal colic. It increases in prominence as the pain progresses, and often waves of peristalsis can be seen to pass across the abdomen. It subsides with the cessation of pain. It is significant and diagnostic if present, but not nearly as frequent as the colicky pain and borborygmus.

Audible sounds of borborygmus are synchronous with intestinal colic and visible peristalsis. They increase in intensity, finally ending in a loud metallic sound with the cessation of colic and peristalsis. It may be audible to the patient or heard by auscultation. Pain and borborygmus are distinctive of obstruction and not commonly found in other forms of abdominal colic. Lead poisoning produces abdominal cramps or colic with localized bowel spasms which might be confusing. In advanced obstruction, pain and peristalsis may be absent and the abdomen silent, because of distention and paralysis of the bowel.

These three clinical entities that constitute the "big three" of obstruction of the small bowel are always present and are usually not appreciated sufficiently early.

Vomiting is of significance when it occurs with abdominal colic and its associated symptoms. With the first onset of obstruction, vomiting may occur immediately as a reflex response. It is a response to the initial shock and not regurgitant from progressive bowel distention.

Early regurgitant vomiting occurs only in high bowel obstruction and not in low small bowel obstruction. Here it is simply of a reflex nature. It is a protective action of nature to put the bowel at rest and to empty it of its accumulated contents. This may even occur in colon obstruction as a reflex physiologic response. It is of greatest diagnostic significance when associated with spasmodic diffuse abdominal colic. At any rate, regurgitant vomiting is associated with progressive proximal bowel distention, and in low bowel obstruction it takes a variable period of time, depending on the level of the obstruction.

High bowel obstruction produces early prostration, profuse emesis, dehydration and severe colicky pain in the upper part of the bowel. There is great loss of fluids and electrolytes, and the urinary output is scanty. Distention is minimal.

The passage of gas or the evacuation of the bowels, either spontaneously or by enema, may occur and give a false sense of security as bowel patency. This has been emphasized by Wangensteen and

Goehl.[1] It is present often in complete bowel obstruction and is reflex in character and another example of nature attempting to put the bowel at rest. It must be remembered that there is nothing physiologically or anatomically wrong with the bowel distal to the obstruction, and it may function in an evacuating capacity with or without stimulation. Therefore, one should not be deceived at the appearance of an apparent good omen.

External evidence of strangulation is positive indication of obstruction, and the presence of scars of a previous abdominal operation or abdominal distention are significant when associated with the clinical symptoms of obstruction. Distention in simple obstruction is symmetric but in strangulated obstruction it is more localized and is most apparent over the loop of distended bowel.

External strangulation needs no comment. It is an immediate surgical "must" and only by this procedure can one be sure of bowel viability, and only then are adequate steps taken to prevent a recurrence. I have no sympathy with the nonsurgical reduction of strangulated hernia, because I am sure that, if it is successful, nothing has been done to prevent a recurrence and the viability of the reduced bowel is uncertain.

Obstructions within the abdomen, hidden from view, may be simple or strangulated, a point of the greatest immediate importance from the point of view of urgent surgical intervention.

A fairly accurate differential diagnosis can be made, and a decision for or against immediate operation in advanced obstruction can be determined by a properly interpreted roentgenogram of the abdomen. In one type, the simple type of obstruction, where the blood supply to the bowel is not impaired, adequate time can be taken for physiologic rehabilitation and bowel decompression, while in the strangulated type gangrene and peritonitis are imminent and operation can not be delayed. By this procedure of a roentgenogram of the abdomen, the distribution and pattern of the gas in the small intestinal tract can be determined.

It must be remembered that gas can be demonstrated normally in the stomach and colon but not in the small intestine. It is present in the small bowel, but is so emulsified with the liquid content that it does not show on a plain radiogram. Only in newborn infants can gas be seen by radiographic study in the small intestine without obstruction. It otherwise indicates some neurogenic or dynamic interference with the continuity of the small bowel, and the pattern assumed by the distended loops indicates in a large measure the nature of the obstruction.

---

1. Wangensteen, O. H., and Goehl, R. O.: Evaluation of the Expulsion of Enemas as a Criterion of Intestinal Obstruction, Arch. Int. Med. **46**:669-679 (Oct.) 1930.

Schwarz and Case [2] early in the century called attention to the significance of gas observed in the small intestines by a roentgenogram. Many radiologists have described characteristic gas patterns in obstruction of the small bowel and have emphasized differential radiologic diagnostic criteria between simple and strangulated obstruction. Lockwood [3] placed great significance on the visibility of the valvulae conniventes as a differential aid in distinguishing between simple and strangulated obstruction. It must be remembered that these markings are more apparent in the upper part of the small bowel than in the lower and that they may still be observed early in strangulated obstruction or when the strangulation is incomplete.

The roentgenogram is the only means by which one can make an early diagnosis of small bowel obstruction and can determine accurately the type of obstruction present.

Gas can be demonstrated in the small bowel within a few hours after obstruction has occurred, and the pattern it assumes in relation to the long axis of the body and the presence or absence of valvulae conniventes are the most significant points in differential diagnosis. In simple obstruction the proximal bowel distends, coil by coil, and gradually assumes a transverse relationship to the long axis of the body, and the valvulae conniventes can be seen. When the obstructed bowel assumes this characteristic stepladder pattern and shows this anatomic configuration, it can be said with relative certainty that the blood supply is not in jeopardy and that adequate time can be taken to intubate the intestines, deflate them and rehabilitate the patient. An operation can be done, then, at a later date, as an elective procedure, when the patient is in an anatomic and physiologic state for operation. Enterostomy is not indicated; it is ineffective and dangerous. This deferred operative treatment applies only to the cases of advanced obstruction, as all patients with early indications should be operated on immediately.

If this pattern of bowel distention is observed to be associated with an inflammatory process, it will subside as the inflammation recedes, and operation for obstruction will not be necessary. I have seen this on many occasions, and even when it has been observed early, I have used intestinal intubation with satisfactory results.

On the contrary, if the distended loop of bowel assumes no definite pattern, is found distended in an irregular manner and is darker than in the previous type and if the valvulae conniventes are not seen or are poorly visible, it may be said that the obstruction is one of a strangulated

2. Case, J. T.: Roentgen Studies After Gastric and Intestinal Operations, J. A. M. A. **65**:1628-1634 (Nov. 6) 1915.

3. Lockwood, I. H.: The X-Ray Examination in Ileus and Intestinal Obstruction, J. Arkansas M. Soc. **37**:225-258, 1941.

type. The blood supply is damaged, and immediate operation is imperative. Efforts toward complete physiologic rehabilitation must be deferred until after operation. It will be noted, as has been mentioned by Wangensteen,[4] that bowel distention proximal to a strangulated loop of bowel is slow to develop and is rarely seen until strangulation has been present for a long time and until, probably, peritonitis has occurred.

The two patterns of simple bowel obstruction and strangulation rarely occur on the same roentgenogram. If they do occur, when there is an irregular loop, dark in color and without valvulae conniventes, the bowel must be considered as strangulated and immediate operation is imperative.

Thus, the types of extrinsic bowel obstructions are two: simple and strangulated. In the simple type, the bowel becomes obstructed by angulation or constriction of an adhesive band or from an adjacent inflammatory process and the viability of the bowel is not in danger. In strangulated obstruction, or volvulous type, the blood supply is impaired and unless quickly released will result in gangrene, rupture and peritonitis.

The roentgen pattern in ileus is that of a uniform distention of the entire intestinal tract, including both small and large bowel. The small intestines assume a transverse position, and the colon distends, forming a framelike pattern around the distended small intestines. This is a valuable observation in the consideration of postoperative abdominal distention. When seen it is not dynamic in character, but probably neurogenic. It sometimes follows trauma or extra-abdominal surgical intervention.

A roentgenogram of the abdomen will give this information to a surprising degree of accuracy. Since 1940 I have correlated clinical and roentgen diagnoses with the operative observations and have found them to be dependable and accurate when properly interpreted by a competent radiologist. I reported, in 1945, from the Research Clinic, the results in 47 cases followed by this procedure.[5] In 6 cases there were inflammatory simple obstructions which were deflated by intestinal intubation without operation, and treated by conservative measures without operation. In 29 cases there were simple adhesive obstructions, part of which were operated on immediately and the remainder treated by intestinal intubation and rehabilitation, followed by subsequent operative release of the nonstrangulating obstruction.

---

4. Wangensteen, O. H., and Lunch, F. W.: Evaluation of X-Ray Evidence as Criteria of Intestinal Obstructions, Proc. Soc. Exper. Biol. & Med. **27**:674-676, 1930.

5. Hunt, C. J.: Early Diagnosis and Management of Small Intestinal Obstruction, Surgery **19**:237-250, 1946.

In 12 cases the roentgenogram showed the pattern of gas distention, characteristic of strangulated obstruction, and operation was done immediately. Resection was done in 4 instances, with recovery.

ILLUSTRATIVE CASE REPORTS

One case in which the condition was of extremely long duration is worthy of mention, as it illustrates the wisdom of conservative measures as contrasted to secondary abdominal operation associated with an acute process and its attending consequences.

Case 1 illustrates the value of conservative decompressive measures.

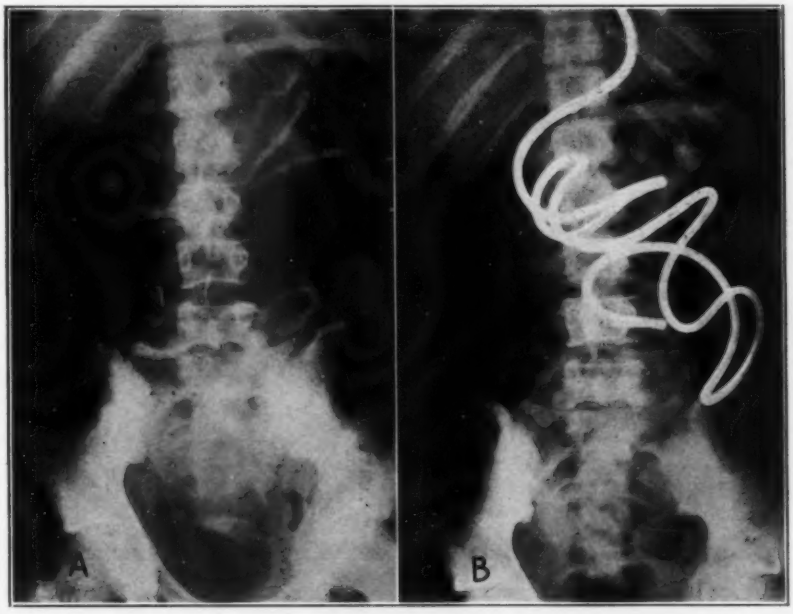

Fig. 1 (case 1).—*A*, simple obstruction secondary to operation for appendical abscess, showing transverse position of distended small intestines. Decompression by Miller-Abbott tube. *B*, recovery after subsidence of infection.

CASE 1.—W. H., a 16 year old boy, presented the picture of an acute abdominal condition of several days' duration. The abdomen was boardlike, but not distended; the pulse was fast, the temperature was elevated 3.5 degrees and the white blood cell count was 20,000. The patient showed no evidence of pneumoperitoneum. Because it had been several days since the onset of acute process and because of the poor condition of the patient, conservative measures were adopted. A mass developed, and subsequently a large appendical abscess was surgically drained and the appendix removed. Convalescence was stormy but progressive until the nineteenth postoperative day, when abdominal pain, periodic and cramplike in character, developed. Diffuse abdominal colic was followed by vomiting. Obstruction was suspected, and a roentgenogram of the abdomen (fig. 1 *A*) showed rather extensive distention of the small bowel. A diagnosis of an inflammatory obstruc-

tion of the small intestine was made. The use of the Miller-Abbott tube (fig. 1 *B*), adequate fluids, limited isotonic sodium chloride solution, dextrose and blood transfusions resulted in complete recovery after a secondary abscess spontaneously drained through the operative incision. After forty days of hospitalization, convalescence was complete and the patient was discharged.

Strangulated obstruction showing a dark distended loop of bowel is well illustrated by the following history, case 2, which shows the pattern of intestinal gas in a middle-aged woman (fig. 2 *A*).

CASE 2.—S. B., a middle-aged woman, had previously had a total hysterectomy for a fundal carcinoma. The roentgen diagnosis was a strangulated obstruction of the small intestine, without proximal bowel distention, and immediate operation was advised. The possibility that obstruction was due to metastatic cancer was considered. At operation an obstruction with volvulus and gangrenous bowel, adhesive and benign in character, was found. Resection with side to side anasta-

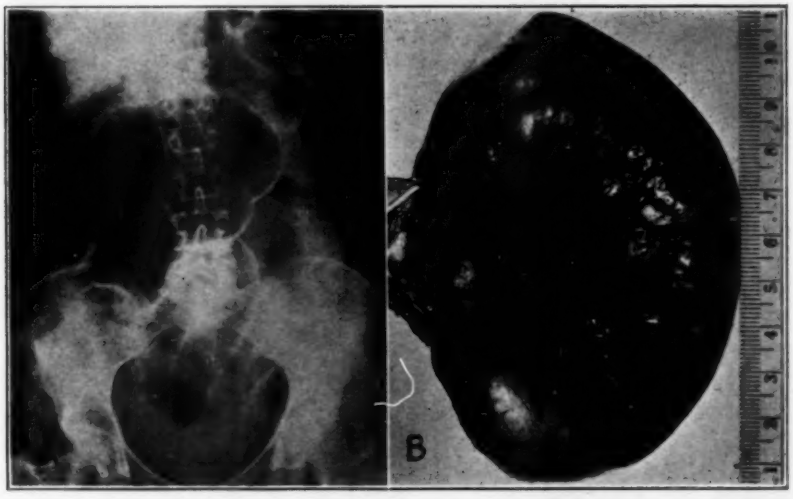

Fig. 2 (case 2).—*A*, irregular pattern of gas in distended bowel characteristic of loop obstruction. Valvulae conniventes are not seen. *B*, resected specimen of gangrenous bowel.

mosis was followed by normal convalescence. The clinical symptoms and the roentgenogram indicated a strangulated obstruction, which demanded immediate operation. The operative observations confirmed this conclusion. The resected specimen is shown in figure 2 *B*.

Mesenteric thrombosis is in reality an obstruction, since peristalsis is abolished and bowel gangrene is imminent. The symptoms are different, as acute sudden pain may usher in the catastrophe. The roentgenogram shows gaseous distribution in the small bowel tract. With sudden onset, continuation of the pain and a gaseous distribution in the small intestines, abdominal exploration should be done. Segmental resection may be possible, but too often the entire supply of the superior mesenteric artery is involved and nothing can be done.

Fig. 3 (case 3).—Dilatation of small bowel, with valvulae conniventes well demonstrated and characteristic pattern of simple small bowel obstruction. The patient had a superior mesenteric embolus.

Fig. 4.—Thrombus in stem superior mesenteric artery.

CASE 3.—Mrs. H., ten days after thyroidectomy for long-standing hyperthyroidism and mild cardiac decompensation with auricular fibrillation, on the eve of her departure from the hospital, was suddenly seized with severe persistent pain in the abdomen. A roentgenogram of the abdomen showed the small bowel to be distended, and at operation the entire bowel and right colon were black. Subsequently, at autopsy, a thrombus was seen plugging the superior mesenteric artery. The origin probably was an embolus of cardiac origin. Figures 3 and 4 show the distention of the small intestines and the autopsy specimen of the involved segment of the superior mesenteric artery.

In patients of advanced years, in the absence of previous abdominal operation or acute localizing signs, the possibility of a colonic lesion must be given careful consideration. This can be most effectively determined with a barium sulfate enema. Such a lesion being excluded, some variety of small bowel obstruction must be considered.

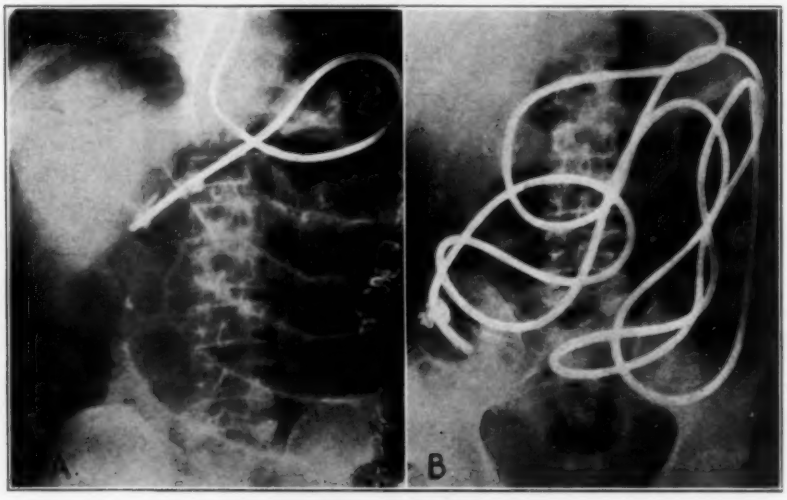

Fig. 5 (case 4).—*A*, roentgenogram of decidedly distended small intestines assuming transverse position and showing valvulae conniventes. Complete decompression by Miller-Abbott tube. *B*, operative recovery after removal of appendix in subsiding acute stage subsequent to bowel decompression.

A simple type of obstruction, showing the characteristic pattern of proximal bowel distention assuming the transverse position with visible valvulae conniventes, is well illustrated in case 4 (fig. 5).

CASE 4.—A man, aged 73 years, presented a three day history of slight abdominal pain and progressive abdominal distention. There had been no vomiting. There was increasing difficulty in bowel evacuation, and none had occurred for five days. No operative abdominal incisions were present. A roentgenogram of the abdomen showed distention of the small bowel (fig. 5 *A*) and some excess amount of gas in the colon. A barium sulfate enema showed no colonic obstruction. A Miller-Abbott tube was passed successfully and the small bowel deflated (fig. 5 *B*). A mass developed in the right iliac fossa, and at operation a gangrenous appendix with abscess was seen. Recovery was uneventful.

INTESTINAL INTUBATION

Although the Miller-Abbott tube has fallen into disrepute in many sections of the country, it is felt to be beneficial and can be applied in selected cases of intestinal obstruction.

The indications for the use of the Miller-Abbott tube in small intestinal obstructions are very definite. In the Research Clinic and Hospital, the procedure of passing the tube is the function of the radiologic department. The department voluntarily has assumed this obligation and has had excellent success.

The tube is indicated in simple adhesive obstruction with extensive distention of the proximal part of the bowel. It is valuable in early obstruction of a similar nature to prevent postoperative ileus and to hasten restoration of an already partially distended bowel. It is contraindicated as a preoperative therapeutic measure in loop obstruction, in volvulus or in strangulated obstruction where the viability of the bowel is in jeopardy. It may be valuable as a postoperative agent to prevent further distention and to hasten restoration of bowel tone.

The Miller-Abbott tube is used in the nonsurgical treatment of those obstructions due to localized inflammatory processes. It is likewise used as an adjunct in part of the operative cases, either as a preoperative or postoperative measure. Clinical and roentgenographic evidence of postoperative obstruction of the small bowel has frequently led to recovery by decompression with the Miller-Abbott tube without operation.

SUMMARY

Attention is called to the early clinical manifestations of obstruction of the small bowel. Emphasis is placed on the character of the colic and other accompanying signs. The clinical insignificance of evacuation of the colon is mentioned. The value of a roentgenogram of the abdomen as a differential diagnostic measure in distinguishing the types of obstruction is strongly emphasized. In advanced simple obstruction intestinal decompression is urged prior to surgical treatment. In strangulated obstruction immediate operation is imperative. The indications for the use of the Miller-Abbott tube are mentioned.

POTASSIUM METABOLISM IN THE IMMEDIATE POSTOPERATIVE PERIOD

ROBERT E. L. BERRY, M.D.

VIVIAN IOB, Ph.D.

AND

KENNETH N. CAMPBELL, M.D.

ANN ARBOR, MICH.

MANY studies on the behavior of body fluids have led to an increasing interest in the subject of cellular electrolytes and the transfer of cations across the cell barrier. Losses of the main cellular cation, potassium, in "excess" of nitrogen have been reported in fasting,[1] dehydration,[2] diabetic coma[3] and diarrhea.[4] Furthermore, partial replacement of deficits of cell potassium by extracellular sodium have been effected in animals[5] on low potassium–high sodium diets, in animals treated with desoxycorticosterone[6] and in patients with alkalosis[7] and infants with diarrhea.[8] The administration of potassium as well

From the Department of Surgery, the University of Michigan, Ann Arbor.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

This work was aided in part by a grant from the John Harper Seeley Fund for Medical and Surgical Research.

1. Benedict, F. G.: A Study of Prolonged Fasting, Publication 203, Carnegie Institute of Washington, 1915.

2. Elkinton, J. R., and Winkler, A. W.: Transfers of Intracellular Potassium in Experimental Dehydration, J. Clin. Investigation **23**:93-101 (Jan.) 1944.

3. Atchley, D. W.; Loeb, R. F.; Richards, D. W., Jr.; Benedict, E. M., and Driscol, M. E.: On Diabetic Acidosis: A Detailed Study of Electrolyte Balances Following the Withdrawal and Reestablishment of Insulin Therapy, J. Clin. Investigation **12**:297-326 (March) 1933.

4. Butler, A. M.; McKhann, C. F., and Gamble, J. L.: Intracellular Fluid Loss in Diarrheal Disease, J. Pediat. **3**:84-92 (July) 1933.

5. Miller, H. C., and Darrow, D. C.: Relation of Muscle Electrolyte to Alterations in Serum Potassium and to the Toxic Effects of Injected Potassium Chloride, Am. J. Physiol. **130**:747-758 (Oct.) 1940.

6. Ferrebee, J. N.; Parker, D.; Carnes, W. H.; Gerity, M. K.; Atchley, D. W., and Loeb, R. F.: Certain Effects of Desoxycorticosterone: The Development of "Diabetes Insipidus" and the Replacement of Muscle Potassium by Sodium in Normal Dog, Am. J. Physiol. **135**:230-237 (Dec.) 1941.

7. Gamble, J. L.; Fahey, K. R.; Appleton, J. E., and MacLachlan, E. A.: Congenital Alkalosis with Diarrhea, J. Pediat. **26**:509-518 (June) 1945. Darrow, D. C.: Congenital Alkalosis with Diarrhea, ibid. **26**:519-532 (June) 1945.

8. Darrow, D. C.: The Retention of Electrolyte During Recovery from Severe Dehydration Due to Diarrhea, J. Pediat. **28**:515-540 (May) 1946.

as sodium has been found to have therapeutic value in some of these conditions.

In this paper data of potassium and nitrogen losses have been analyzed from a large number of infusion studies in control subjects and patients undergoing mild and traumatizing operations (herniorrhaphies and combined abdominoperineal resections for carcinoma of the rectum). Urinary outputs of potassium and nitrogen were determined for 4 or 5 six hour periods for the first thirty hours postoperatively. Infusions of sodium chloride solutions and of 5 per cent dextrose were

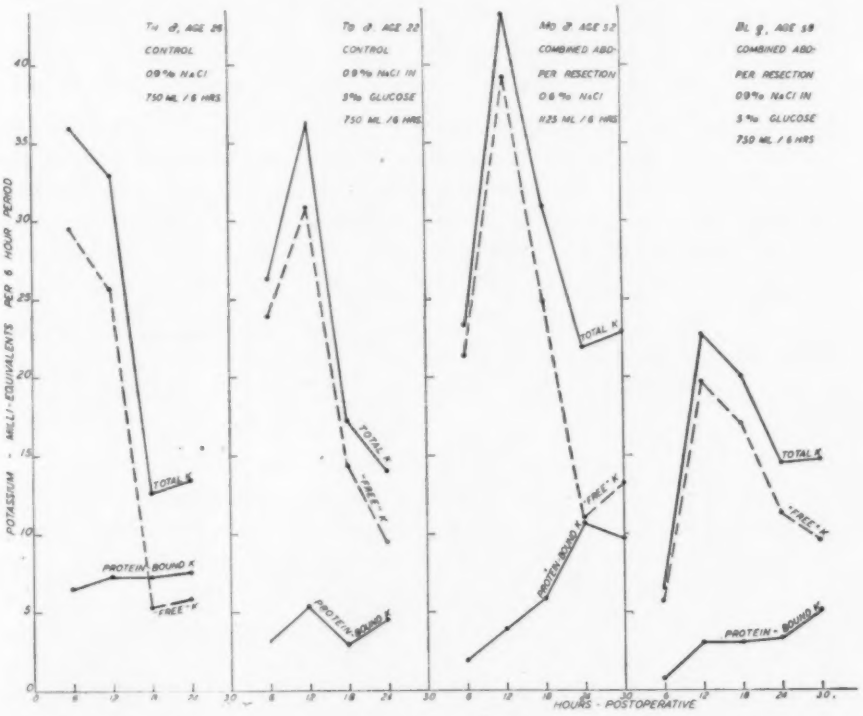

Fig. 1.—The renal excretion of total potassium, protein-bound potassium and "free" potassium during the intravenous infusion of sodium chloride solutions. Controls Th and To received 3,000 ml. of 0.9 per cent sodium chloride and 0.9 per cent sodium chloride in 5 per cent dextrose during twenty-four hours. Patients Mo and Bl received 5,625 ml. of 0.6 per cent sodium chloride and 3,750 ml. of 0.9 per cent sodium chloride in 5 per cent dextrose, respectively, in the thirty hour period following combined abdominoperineal resection.

administered at approximate rates of 250 ml. per hour for the infusions of 750 ml. and 400 ml. per hour for the larger volumes. The procedure and the chemical methods [9] outlined in an earlier paper were followed.

9. Coller, F. A.; Iob, V.; Kalder, N. B.; Vaugan, H. H., and Moyer, C. A.: Translocation of Fluid Produced by Intravenous Administration of Isotonic Salt Solutions in Man Postoperatively, Ann. Surg. **122**:663-677 (Oct.) 1945.

Urinary losses of potassium and nitrogen of 5 control subjects (healthy young men), 4 patients submitted to inguinal herniorrhaphy and 6 patients undergoing one stage abdominoperineal resections are presented in figures 1, 2 and 3. The potassium to nitrogen ratio used for estimating protein-bound potassium was 2.75.[10]

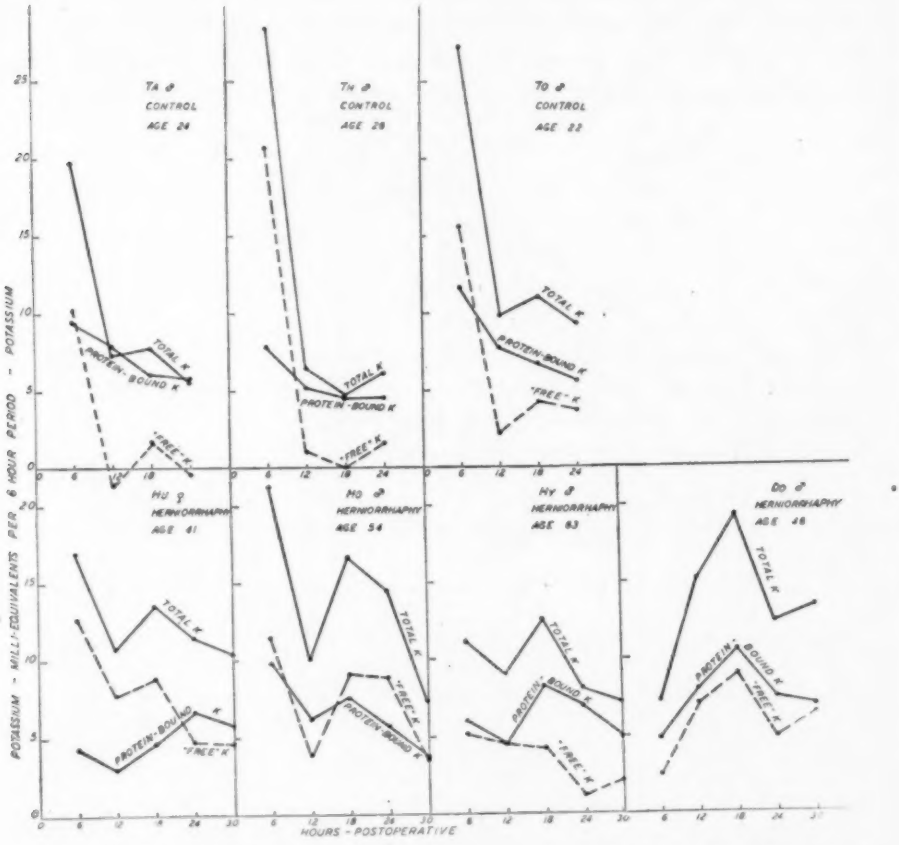
POTASSIUM - MILLI-EQUIVALENTS PER 6 HOUR PERIOD - POTASSIUM
HOURS - POSTOPERATIVE
Ta ♂ CONTROL AGE 24
Th ♂ CONTROL AGE 26
To ♂ CONTROL AGE 22
Hu ♀ HERNIORRHAPHY AGE 41
Ho ♂ HERNIORRHAPHY AGE 54
Hy ♂ HERNIORRHAPHY AGE 63
Do ♂ HERNIORRHAPHY AGE 46
TOTAL K
PROTEIN-BOUND K
"FREE" K

Fig. 2.—The renal excretion of total potassium, protein-bound potassium and "free" potassium during intravenous infusion of 5 per cent dextrose, 750 ml. per six hour period. Controls Ta, Th and To received 3,000 ml. in a twenty-four hour period. Patients Hu, Ho, Hy and Do received 3,750 ml. during thirty hours after herniorrhaphy.

10. Katz's analyses of human muscle gave values of 2.15 milliequivalents of potassium per gram of nitrogen, a figure supported by the ratios found during the last six periods in the fast of Benedict's subject.[1] In analyses of human fetuses (Iob, V., and Swanson, W. W.: Mineral Growth in the Fetus, Am. J. Dis. Child. **47**:302-306 [Feb.] 1934) values of 3.0 to 2.1 (term fetus) have been reported. However, Reifenstein[16] has calculated the ratio to be 2.7 in the early part of the fast, a figure checked by the anabolism induced by testosterone (2.77). Therefore, the use of the higher factor in the early postoperative period is indicated.

Figure 1 presents the renal excretion of total potassium, protein-bound potassium and "free" or excess potassium during intravenous infusion of sodium chloride solutions in 2 young healthy subjects, Th and To, and 2 patients undergoing combined abdominoperineal resection, Mo and Bl. Input of sodium was the same for each subject. For control Th, the peak of potassium output was reached at the end of six to twelve hours. It then fell rapidly, and at the end of twenty-four hours, protein-bound potassium represented more than half of the potassium loss. During the entire period, however, the free potassium represented 73 per cent of the total loss. Similarly, control To, whose handling of the salt load resulted in mild hypertonicity, reached the peak of potassium output at the end of twelve hours. As in the first control, potassium excretion then fell rapidly.

Patients Mo and Bl excreted more water than salt, and present comparable stages of hypertonicity. However, practically twice as much potassium was lost by Mo. Peak excretion was reached at the end of twelve hours in both studies, and at twenty-four hours, free potassium represented 81 and 84 per cent of the total. Postoperative oliguria, with retention of both nitrogen and potassium, occurred in the six to twelve hour periods in both patients.

Figure 2 shows the renal excretion of potassium during the intravenous infusion of 5 per cent dextrose in 3 control subjects and 4 patients submitting to herniorrhaphy.

The 3 controls present essentially the same picture: highest excretion of total potassium at the end of six hours, which falls off rapidly during the second period. Protein-bound potassium now represents practically all of the total potassium excreted, in spite of the dehydrating tendency of 5 per cent dextrose solution (table). Controls Th and To had previously received sodium chloride solutions (fig. 1). The response of potassium excretion to load of salt was twice the excretion with dextrose solution.

All 4 patients submitting to herniorrhaphy present renal response to infusion differing from that of the controls. The immediate peak response is absent. In only 1 instance (Hy) does protein-bound potassium represent almost all of the total potassium. However, at thirty hours, protein-bound potassium exceeds free potassium.

The renal excretions of potassium of 4 patients undergoing one stage abdominoperineal resection are shown in figure 3. The contrast between excretion of potassium in the younger patients (Sc and He) and the older patients (Ro and Bi) is pronounced. Muscle stores of potassium may be more easily mobilized in the younger patients and may account for the larger losses. All patients submitting to the more traumatizing operation show a lag in reaching maximal potassium

outputs, a result of temporary renal suppression in the early postoperative hours. Free potassium outputs remain high, averaging 73 per cent, higher than the percentages found in the controls and in patients after herniorrhaphy who received dextrose.

The table summarizes the observations for load of water and excretion of potassium and nitrogen for all subjects at the end of twenty-four hours. Groups I and II may be judged hypertonic, group III isotonic and group IV hypotonic with respect to handling of the

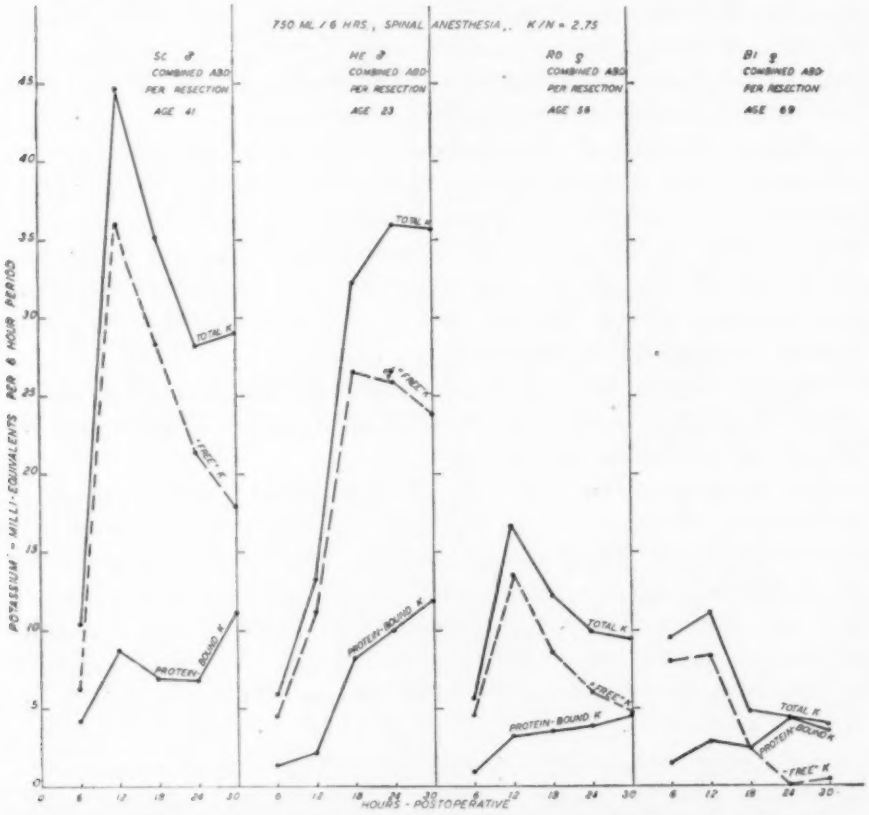

Fig. 3.—Renal excretion of total potassium, protein-bound potassium and "free" potassium during intravenous infusion of 5 per cent dextrose after combined abdominoperineal resection. Patients Sc, He, Ro and Bi received 750 ml. per six hour period for five periods.

various infusates. However, free potassium was lowest (average: 42 per cent) for the control group, which lost weight during the infusion of 5 per cent dextrose. It was practically the same (50 per cent) for the group submitting to herniorrhaphy; free potassium was increased for the group undergoing the more traumatizing operation and/or receiving sodium chloride solutions.

COMMENT ON RESULTS

Negative potassium balances observed in these studies are of interest with respect to the following questions: (1) Why are the potassium losses relatively high during the early postoperative hours? (2) What is the cause of the discrepancy between outputs of potassium and nitrogen? (3) To what extent may the vital functions of the cell be compromised by the removal of such quantities of physiologically important potassium?

In his studies of fasting subjects, Gamble [11] explained "excess" potassium in the urine on the basis of loss of cell water through

Renal Excretion of Potassium and Nitrogen, Twenty-Four Hours After Infusion of Sodium Chloride Solutions and 5 per Cent Dextrose in Control Subjects and Patients Postoperatively

Group	Subject	Infusate	Load Water, Ml.	Renal Nitrogen, Gm.	Total mEq.	Renal Potassium Protein-bound, mEq.	"Free" mEq.	"Free" Percentage of Total
I	Th (control)	0.9% Sodium chloride	157	10.46	109	29	80	73
I	To (control)	0.9% Sodium chloride *	1,317	5.86	108	16	92	85
I	Mo (combined)	0.6% Sodium chloride	838	8.27	119	23	96	81
I	Bl (combined)	0.9% Sodium chloride *	1,451	3.66	64	10	54	84
II	Th (control).	5% Dextrose	−1,637	8.01	46	22	24	52
II	To (control)	5% Dextrose	−1,028	11.61	58	32	26	45
II	Ta (control)	5% Dextrose	−346	10.65	40	29	11	28
III	Do (herniorrhaphy)	5% Dextrose	−18	11.15	54	31	23	43
III	Hy (herniorrhaphy)	5% Dextrose	81	9.35	41	26	15	37
III	Ho (herniorrhaphy)	5% Dextrose	892	10.64	63	29	34	54
III	Hu (herniorrhaphy)	5% Dextrose	−110	6.80	53	19	34	64
IV	He (combined)	5% Dextrose	1,424	7.06	88	19	69	78
IV	Se (combined)	5% Dextrose	596	10.42	119	29	90	76
IV	Ro (combined)	5% Dextrose	818	4.25	44	12	32	73
IV	Bi (combined)	5% Dextrose	1,594	3.97	30	11	19	63

* 0.9 per cent sodium chloride in 5 per cent dextrose.

glycogenolysis and resulting loss of potassium. Byrom,[12] working with diabetic persons, observed that the potassium outputs of patients receiving dextrose in intravenous infusions were not significantly decreased until potassium was administered parenterally. In the current experiments, dextrose was found to be ineffective in decreasing the negative balances of potassium observed during the first six to twelve hours postoperatively in patients and in controls. Furthermore, a

11. Gamble, J. L.; Ross, G. S., and Tisdall, F. F.: The Metabolism of Fixed Base During Fasting, J. Biol. Chem. **57**:633-695 (Oct.) 1923.

12. Byrom, F. B.: The Effect of a Diet of Pure Glucose on the Fluid Balance of the Body, Clin. Sc. **1**:245-250 (Nov.) 1934.

loss of body weight may accompany dextrose and water feeding, as these substances are not capable of totally protecting extracellular fluid.

It is highly probable that anesthesia and operative trauma are, in part, responsible for the sustained high potassium output in patients. Stewart and Rourke [13] expressed the belief that the excess potassium diuresis observed in their patients postoperatively was due, in some degree, to the operative trauma and the anesthesia. Operative trauma may be reflected in the increased output of potassium by patients He and Sc. On the other hand, decreased output of potassium and nitrogen in the older patients, especially true of elderly women, may be indicative of decreased protein synthesis as a primary difficulty and, therefore, poorer stores for the catabolism incident to fasting.

It is conceivable that blood loss incurred in operation may be a contributing factor to augmented potassium excretion. Hemorrhage has been demonstrated to cause a sharp increase in serum potassium in animals. Thaler,[14] working with cats, observed that a loss of 40 to 50 per cent of the blood volume increased the serum potassium concentrations to high levels.

These factors do not suffice to explain the potassium diuresis observed in controls and patients submitting to less traumatizing operations. Many previous discussions of the problems of body water and electrolyte have assumed that the cell membranes are practically impermeable to cations. This surmise is now known to be incorrect. The initial infusion of fluids intravenously, with or without salt, apparently necessitates adjustments between the intracellular and extracellular fluid compartments because of a lag in adjustment of renal function. Darrow and Yannet [15] have pointed out that because the adjustment of the kidney to loads of water and salt is slow, a more rapid method of adjustment is afforded by a shift of water from the intracellular to the extracellular compartment. Furthermore, so-called isotonic solutions may not be necessarily isotonic when infused, as the various compartments of body water possess considerable variability in respect to content of normal electrolyte and water. It is therefore felt that the chief factor in pronounced potassium diuresis during the first six to eighteen hours of these studies is the necessity of adjustment of the compartments of body water to fluids given parenterally. It would appear that the greater part of this adjustment is at the expense of the intracellular fluid. The rapid decline in outputs of potassium

13. Stewart, J. D., and Rourke, G. M.: The Effects of Large Intravenous Infusions on Body Fluid, J. Clin. Investigation **21**:197-205 (March) 1942.

14. Thaler, J. I.: Evidence of Permeability of Tissue to Potassium, Proc. Soc. Exper. Biol. & Med. **33**:368-371 (Dec.) 1935.

15. Darrow, D. C., and Yannet, H.: Changes in Distribution of Body Water Accompanying Increase and Decrease in Extracellular Electrolyte, J. Clin. Investigation **14**:266-275 (March) 1935.

during the early phase, particularly in the controls receiving dextrose solution, suggests that a more satisfactory adjustment between parenteral intake and renal output has been accomplished, since subsequent infusions do not necessarily cause a return to the initial high values.

Although these studies have not been carried out beyond thirty hours, greatest loss of potassium apparently does occur in this period. Reifenstein [16] observed greater negative balances of potassium than nitrogen in patients during the catabolic phase of the first four postoperative days. During the subsequent four days, however, there was positive potassium balance, while nitrogen reached equilibrium.

These losses of potassium in the experiments presented here, although large, are only a small fraction of the total intracellular potassium and were not associated with the clinical signs and symptoms of intracellular potassium deficit.[17]

It has been observed that the utilization of various amino acid mixtures administered parenterally in the immediate postoperative post-traumatic period, particularly in young persons who have suffered a damaging event, is not so efficient as it is later in the postoperative recovery period or in eldery asthenic patients. The greater potassium diuresis observed in the younger patients during the early postoperative hours could conceivably contribute to the low coefficient of amino acid utilization by creating a potassium deficit. However, actual loss of potassium does not seem sufficiently large to support such a conjecture.

Although the need of reinforcing with potassium the fluids being utilized parenterally to reestablish water and electrolyte balances in severe diarrhea of infants is now recognized, the necessity of such reinforcement in short term therapy has not been established. There is evidence suggesting that prolonged pyloric obstruction or protracted gastric suction may produce states of severe intracelluar deficit of potassium. Administration of potassium chloride in concentrations to 2 Gm. per thousand milliliters of fluid given parenterally has been recommended as an aid in correction of the electrolyte imbalance. However, the intravenous administration of potassium salts is not without danger, since concentrations above 10 milliequivalents of potassium per liter of extracellular fluid may cause heart block. Evidence of functioning kidneys should be obtained before potassium salts are given intravenously.

SUMMARY AND CONCLUSION

The renal excretion of potassium and nitrogen has been studied in normal young men and in patients undergoing herniorrhaphy and combined abdominoperineal resection for carcinoma of the rectum.

16. Reifenstein, E. C., Jr.: Read before the Conference on Metabolic Aspects of Convalesence, Oct. 15, 1945.

17. Moyer, C. A.: Fluid and Electrolyte Balance, Surg., Gynec. & Obst. **84**: 586-599 (April) 1947.

During the immediate postoperative period, potassium is excreted in the urine in excess of that anticipated from the breakdown of protoplasm. The necessity for the adjustment of the various compartments of body water to the administration of intravenous fluids is considered the most important factor involved. Such adjustment is apparently accomplished at the expense of the intracellular fluid. Other contributing factors may be: (1) anesthesia and trauma of operation, (2) load of sodium chloride, (3) dehydration, (4) hemorrhage at the time of operation and (5) glycogenolysis.

Losses of potassium observed in these patients are large, but constitute only a small fraction of total cellular potassium. Signs of intracellular potassium deficit were not observed.

There appears to be little necessity to reinforce solutions given parenterally with potassium in short term therapy, provided that preoperative preparation has been adequate.

LYMPHATIC SPREAD OF CARCINOMA OF THE BREAST

CLARENCE W. MONROE, M.D.
CHICAGO

IN 1938, an anatomic and pathologic study of the lymphatic spread of carcinoma of the breast was started at the Presbyterian Hospital as a corollary to the work already done by Gilchrist and David on the lymphatic spread of carcinoma of the bowel. Surgical specimens removed by radical mastectomy were washed completely free of blood, fixed in 10 per cent formaldehyde solution for twenty-four hours, and then dehydrated in varying strengths of alcohol from 50 per cent to absolute. The specimens were then cleared in oil of wintergreen. From such cleared specimens all the lymph nodes were removed, their location marked on an appropriate diagram of the thoracic wall and each of the lymph nodes sectioned and examined microscopically. From 1938 through 1942, a total of 87 surgical specimens of carcinoma of the breast were studied in this fashion. These were removed by fifteen different surgeons on the hospital staff, but 68 of the specimens were removed by six surgeons. All specimens covered in the present study were removed as radical mastectomies in instances in which it was anticipated by the surgeon at the time of the operative procedure that a clinical cure might be secured. Some specimens of palliative resections were cleared, but those have been discarded from this study.

It was my impression, when this study was started, that it would be possible by a meticulous study of the location of lymphatic metastases to plan the operation of carcinoma of the breast in a specific way for a lesion located in a particular area of the breast. This has proved true only to a degree. In figure 1, a lesion in the upper outer quadrant coming under the nipple has its only lymph node metastasis in the node usually associated with Rotter's name. Figure 2 shows a primary lesion in the same location, but five lymph nodes were involved out of a total of forty-six. There were many lymph nodes which were completely by-passed in the course of the lymphatic spread. Figure 3 demonstrates that it is possible to find involved lymph nodes high in the axilla, with many intervening nodes free of cancer. Figure 4 further illustrates

---

From the Presbyterian Hospital.

This investigation was aided by a grant from the Otho S. A. Sprague Memorial Fund.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

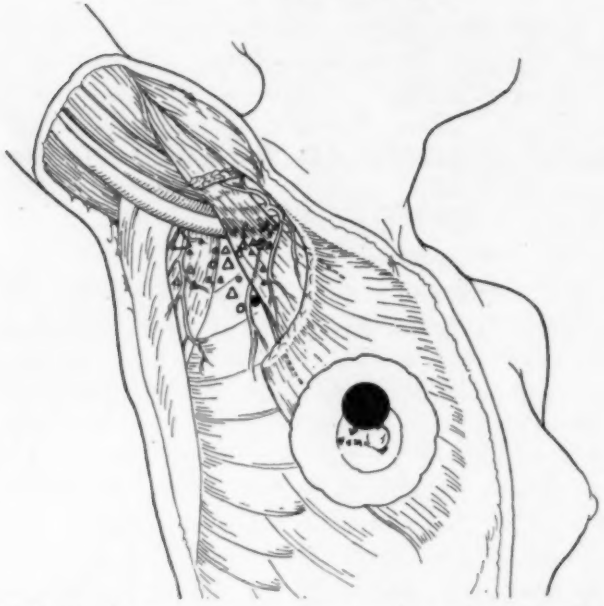

Fig. 1.—Lesion in the upper outer quadrant coming under the areola. Of thirty-four nodes removed there was only one which contained carcinoma. It is located at the point which Rotter first described as being the usual first site of spread of carcinoma of the breast. The patient was well after nine years. The circles are anterior to the plane of the pectoralis minor muscle; the triangles are posterior to the plane of this muscle. Involved nodes are in solid black.

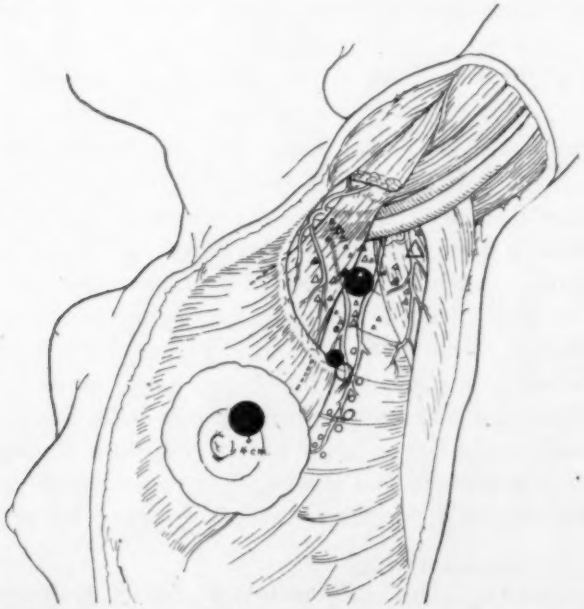

Fig. 2.—Lesion in same location as in figure 1, but five of forty-six lymph nodes contain carcinoma. Many nodes are by-passed in spread of disease. The patient died of carcinoma five years after resection.

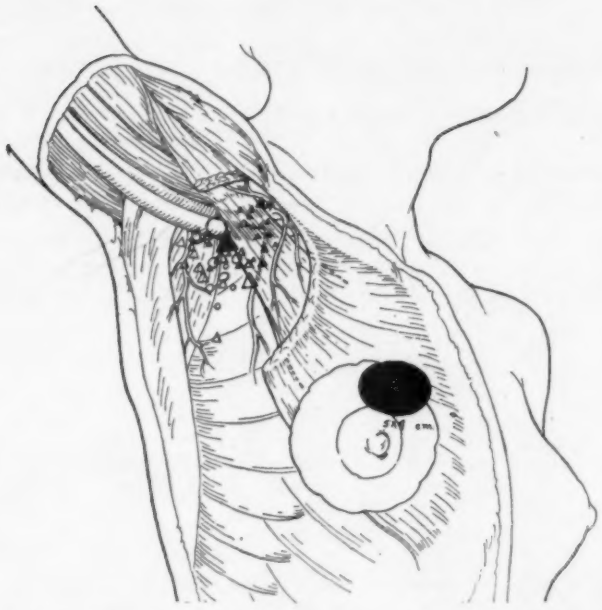

Fig. 3.—Lesion in the upper inner quadrant, but coming under the areola. The upper metastasis was secured only because the surgeon went high under the clavicle to get it. The patient lived one year.

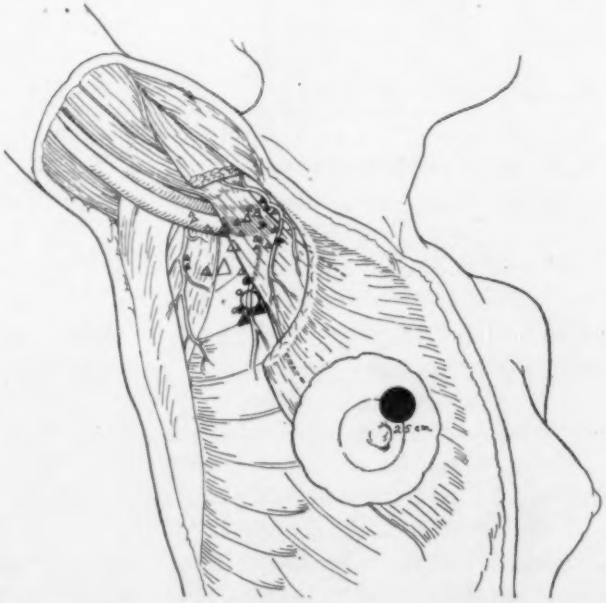

Fig. 4.—Lesion in the same location as in figure 3, and again involved nodes are found high under the clavicle. The patient was alive and well more than six years.

TABLE 1.—*Résumé of 87 Surgical Specimens of Breast Carcinoma, with Number of Lymph Nodes Found, the Number Which Contained Carcinoma and the Period of Known Survival*

Surgeon	No Nodes Involved									One Node Involved *						More Than One Node Involved *					
	Alive and Well			Alive with Disease			Dead			Alive and Well			Dead			Alive and Well			Dead		
	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.	Number of Nodes Found	Number of Nodes Involved	Survival, Yr.
A	20	0	6 4/12	30	0	6 6/12	17	0	3	..	..	.....	..	..	.....	15	2	7 7/12	10	2	3/12
	7	0	6 6/12	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	15	11	1
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	18	7	1
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	12	7	2 1/12
B	21	0	6 6/12	..	..	.....	23	0	5	13	1	6	15	1	2 7/12	34	25	5	59	47	5 6/12
	25	0	6 3/12	..	..	.....	30	0	10/12 †	35	1	7 7/12	34	1	5 6/12	18	4	8	22	10	?
	13	0	7 6/12	..	..	.....	..	..	..	33	1	5	..	..	.....	..	..	.....	34	4	6 4/12
	23	0	8 5/12	..	..	.....	..	..	..	43	1	5	..	..	.....	..	..	.....	35	35	11/12
	..	..	.....	..	..	.....	..	..	..	46	1	5 10/12	..	..	.....	..	..	.....	33	6	1
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	10	2	1 6/12
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	26	6	1 7/12 †
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	26	5	2 7/12 †
C	10	0	7 6/12	..	..	.....	..	..	..	..	..	.....	25	1	? †	26	4	8	42	14	?
	20	0	5 7/12	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	19	7	5 4/12
	21	0	6 2/12	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	34	22	2 2/12 †
	10	0	5 1/12	..	..	.....	..	..	..	..	..	.....	..	..	.....	..	..	.....	29	2	2 †
	21	0	7 8/12																		
	21	0	6																		
	41	0	6 11/12																		
D	27	0	7 9/12	..	..	.....	..	..	..	..	..	.....	32	1	5	19	2	5 8/12	46	5	5
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	46	6	6 9/12	45	34	4/12
E	15	0	6	..	..	.....	..	..	..	51	1	5 1/12	..	..	.....	..	..	.....	34	7	1
	55	0	7 2/12	..	..	.....	..	..	..												
	26	0	7 6/12																		
	42	0	6 1/12																		
F	50	0	6 6/12	..	..	.....	..	..	..	..	..	.....	..	..	....	68	3	6	42	2	1 1/12
	25	0	7	..	..	.....	..	..	..	..	..	.....	..	..	.....	32	8	6	65	11	3
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	35	2	5 4/12	14	4	8/12
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	63	7	5 6/12	46	3	9/12
	..	..	.....	..	..	.....	..	..	..	..	..	.....	..	..	.....	70	6	7	..	..	.....
Others (9)	3	0	5 2/12	..	..	.....	19	0	? †	34	1	8 8/12	28	1	4	16	9	5 7/12	34	25	1 9/12
	28	0	6 6/12	..	..	.....	..	..	..	28	1	5 9/12	46	1	? †	..	..	.....	19	18	? †
	52	0	7	..	..	.....	33	0	5 10/12	..	..	.....	23	1	1 9/12	38	5	5 6/12	24	22	8/12
	..	..	.....	..	..	.....	37	0	? †	..	..	.....	25	1	8 days	..	..	.....	48	3‡	6 10/12
	..	..	.....	..	..	.....	43	0	? †	..	..	.....	..	..	.....	..	..	.....	..	..	.....

* There were no patients in this category alive with disease.
† Untraced beyond the period of years and/or months indicated.
‡ Died of other causes without carcinoma.

the same fact. It becomes clear then that nothing less than the most radical resection of the breast and axillary contents which can be devised can be counted on to remove the disease when lymphatic spread has occurred.

Of the 87 specimens, 31, or 35.6 per cent, did not contain lymph node metastases. Fifty-six specimens, or 64.4 per cent, did contain metastases in the lymph nodes, and of these 16, or 18.4 per cent, contained only one involved lymph node, out of the entire group removed in that particular specimen. The number of lymph nodes removed in these various specimens varied from 3 to 70 in a single specimen. The average for the group was 30.4 lymph nodes per specimen. These nodes varied in size from 0.5 mm. to 50 or 60 mm. in diameter. A total

TABLE 2.—*Average Number of Lymph Nodes Per Specimen Removed by Various Surgeons and the Five Year Recovery Rate of Their Patients*

		Number of Patients			Without Node Metastases		With One Node Involved		With More Than One Node Involved	
Surgeon	Average Nodes per Specimen	Total Number	Untraced	Percentage Free of Disease for 5 Yr.	Number of Patients	Percentage Free of Disease for 5 Yr.	Number	Percentage Free of Disease for 5 Yr.	Number	Percentage Free of Disease for 5 Yr.
A	16	9	0	33	4	50	0	..	5	20
B	28.2	23	4	48	6	67	7	71	10	20
C	28.8	13	4	62	7	100	1	0	5	20
D	35.8	6	0	50	1	100	1	0	4	50
E	38	6	0	83	4	100	1	100	1	0
F	46.8	11	0	63	2	100	0	..	9	55
Others (9)	30.4	19	6	42	7	43	6	33	6	50
Total	30.4	87	18	51.7	31	74.2	16	56	40	37

of 2,643 lymph nodes were sectioned from these 87 specimens. From two to ten sections of each of these nodes were examined microscopically.

A five year follow-up of these 87 patients was completed. Table 1 gives a complete résumé of each of the cases, showing the number of nodes removed, how many were involved with carcinoma and the number of years the patient lived. Only 13 of the 87 remain untraced, for a loss rate of 14.9 per cent. In this paper, each of those untraced patients is considered dead. Forty-five of the original 87 patients, or 51.7 per cent, had remained free of disease five years or more. One patient with recurrence was alive and well on endocrine therapy more than seven years after resection. Forty-one patients were dead of carcinoma, but 7 of these survived five years or more with disease. Of the 31 patients who did not have metastases in their operative specimens, 23, or 74.2 per cent, were free of disease for five years or more. Of those with metastases, there were 22, or 39.3 per cent, who remained free of disease for five years or more.

* There were no patients in this category alive with disease.
† Untraced beyond the period of years and/or months indicated.
‡ Died of other causes without carcinoma.

Table 2 shows the distribution of cases among the six principal surgeons operating in this group and indicates the number of lymph nodes which each of them averaged in their operative specimens. The percentages shown alongside each of the numbers of cases indicate the percentage of these small numbers of patients who were free of disease for the five year period. The same information is shown graphically in figure 5. These numbers are obviously too small to permit any final deduction. However, they do definitely indicate some important things. Surgeon A, who removed an average of sixteen lymph nodes in each specimen, has only 33 per cent of his 9 patients well at the end of five

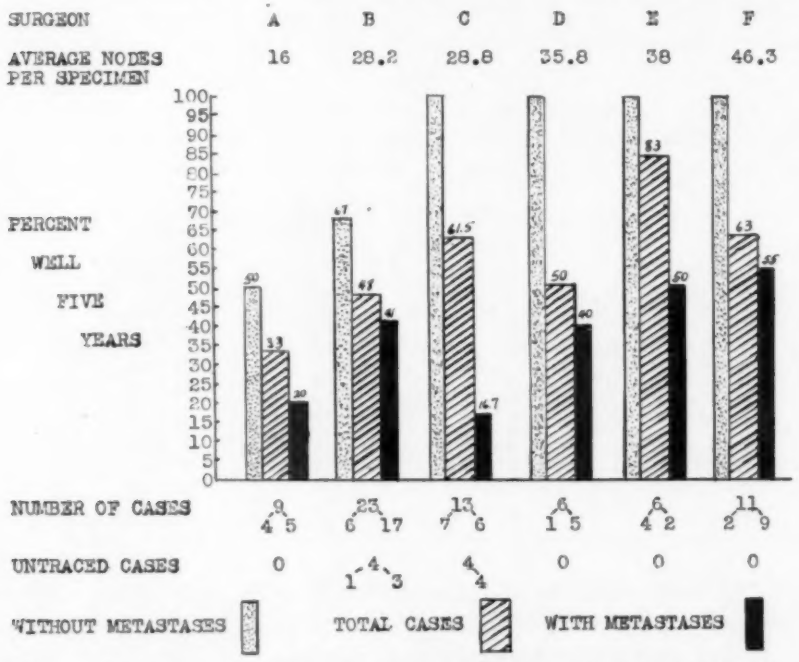

Fig. 5.—Diagrammatic representation of material contained in table 2, showing the recovery rate secured by various surgeons.

years. Each of the other operators in the group has made a somewhat better average, and each has also removed considerably more than sixteen lymph nodes per specimen. Surgeons B and C, removing approximately the same number of lymph nodes per specimen, have a decidedly better five year recovery rate for their entire group than does surgeon A. The most significant features in this table are the figures for surgeon F with 11 cases, 9 of the patients having had more than 1 lymph node involved in each specimen. He secured a recovery rate of 63 per cent. This is not the highest rate, since surgeon E secured an 83 per cent recovery rate for his entire group. However, he had only

6 patients; 4 of these 6 had no lymph nodes involved, and 1 had only one lymph node involved.

This study indicates then that the larger the number of lymph nodes removed in a surgical specimen, the greater are the patient's chances for recovery. This thesis may be analyzed from another point of view. If one were to consider the number of lymph nodes present in each specimen as a criterion of what the patient's chances for recovery would be, rather than who the surgeon was, one should be able to find the same thing.

Fig. 6 indicates what the patient's relative chance for freedom from disease for five years or more was when axillary metastases were present, as measured against the number of lymph nodes removed in the operative specimen. One may see in the first column that if less than thirty lymph

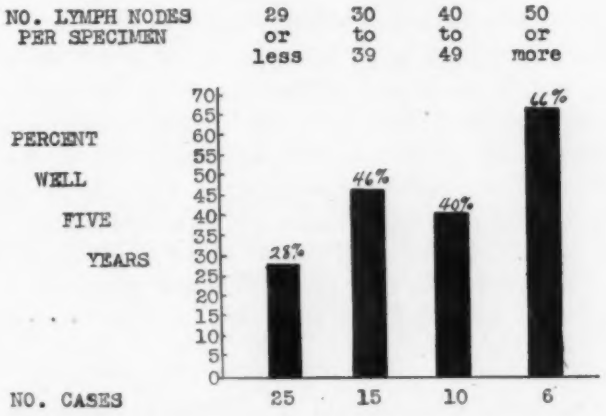

Fig. 6.—Effect on the recovery rate with increased removal of lymph nodes in radical mastectomy; number of patients well five years after operation with axillary metastases present.

nodes were taken in each specimen, only 28 per cent of the patients remained free of disease for five years. If thirty to thirty-nine lymph nodes were taken, 46 per cent of the patients remained well. In the 10 cases in which forty to forty-nine nodes were removed in each specimen, 40 per cent of the patients remained well more than five years, and in the 6 cases in which fifty or more nodes were removed, there was sixty-six per cent freedom from disease for five years.

As stated previously, the number of cases presented here is not sufficiently large to make this point absolutely proved; however, there does seem to be definite pointing in this direction. One may readily ask then, "What is the difference in radical mastectomy that makes the above figures?" In my experience, from examining this group of operative specimens and watching the operative procedures, there is a distinct difference in the surgical procedure in different hands. The

operative specimens which contained the largest number of lymph nodes were removed by sacrificing the medial half of the clavicular portion of the pectoralis major muscle and then immediately passing upward under the clavicle over the vessels and brachial plexus to remove all lymph-node-bearing tissue from this region, carrying the dissection downward and lateralward. The axillary vessels were laid entirely bare of fat and fascia. The entire specimen came to the laboratory then as a single block in which all the tissues were firmly bound to each other. On the other hand, the specimens which contained a small number of lymph nodes on the average contained the axillary tissues hanging by a few fine strands of tissue to the main body of the breast and pectoral muscles. There was not a definite block resection of the axillary tissues with the breast, which most men agree is the preferable procedure in surgical treatment of carcinoma.

SUMMARY

A detailed pathologic study has been made of 87 breast specimens removed at radical mastectomy. The location of the lymph nodes has been accurately charted and each of the lymph nodes examined microscopically. The study indicates that the greater the number of lymph nodes removed in the operative specimen, the greater are the patient's chances for freedom from recurrence. A comparison is made between the work of individual operators and also a comparison between the number of lymph nodes present in each of the different specimens as compared with the patient's remaining free of disease for a five year period.

DISCUSSION

DR. WALTER G. MADDOCK, Chicago: I would like to compliment Dr. Monroe for his excellent paper and to make one point. There is a tendency in various parts of the country to do simple mastectomies or incomplete radical mastectomies for carcinoma of the breast. I cannot see how anyone could defend such treatment after listening to what Dr. Monroe has said this morning. With a radical mastectomy and removal of the greatest possible number of lymph nodes, the patient has the best chance of survival.

EXPERIENCES WITH THE DRAGSTEDT SKIN-COVERED ILEOSTOMY

GEORGE BENTON SANDERS, M.D.
LOUISVILLE, KY.

THE GREAT value of ileostomy in the surgical attack on such diseases as ulcerative colitis and multiple colonic polyposis is often nullified partly or wholly by the refusal of patients to submit to the procedure or, having submitted, to bear with fortitude the abnormal, unpleasant and often painful existence which follows. The most important disadvantage to the patient with an ileostomy is the painful excoriation produced by the leakage of ileal content onto the surrounding skin. Many patients attempt to reduce the amount of excoriating discharge by extreme limitation of food and fluid intake, which is detrimental to the proper dietary management of such conditions as ulcerative colitis. Pain, induced semistarvation and the general messiness of the required toilet greatly lower morale and cooperation and limit social and economic activity.

Improvements in the technic of making the ileostomy and in the construction of suitable appliances [1] have attempted to minimize excoriation and to eliminate the messy protective ointments and cumbersome and unesthetic receptacles which burden the life of the patient with an ileostomy. No combination of these has been entirely satisfactory. The Dragstedt skin-covered ileostomy does so much more to achieve these ends than any other with which I am now familiar that the temerity of reporting an experience based on only 5 cases may, perhaps, be justified.

TECHNIC

The technic of ileostomy to be described was devised by Dr. Lester Dragstedt of Chicago, who, in 1941, reported its use in 4 cases.[2] The

---

Fellow of the American College of Surgeons.

From the Department of Surgery, University of Louisville Medical School, Louisville, Ky.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

1. (*a*) Cattell, R. B.: A New Type of Ileostomy for Chronic Ulcerative Colitis, S. Clin. North America **19**:629, 1939; (*b*) Technic of Total Colectomy for Ulcerative Colitis, ibid. **24**:661, 1944; (*c*) The Care of Ileostomy, Lahey Clin. Bull. **4**:45, 1944. (*d*) Dennis, C.: Ileostomy and Colectomy in Chronic Ulcerative Colitis, Surgery **18**:435, 1945.

2. Dragstedt, L. R.; Dack, C. M., and Kirsner, J. B.: Chronic Ulcerative Colitis: Summary of Evidence Implicating Bacterium Necrophorum as the Etiologic Agent, Ann. Surg. **114**:653, 1941.

method consists, briefly, in fashioning a skin-graft-covered spigot of terminal ileum about 3 inches (7.6 cm.) long in its finished form, which is not unlike a penis and which can easily be fitted with a variety of simple appliances.

The abdomen is opened under local or low spinal anesthesia, through a short, 2 to 3 inch (5 to 7.6 cm.) right rectus muscle-splitting incision placed about 2 fingerbreadths below and to the right of the umbilicus. Proper location of the incision is important for the later fitting of an appliance. A suitable segment of terminal ileum is chosen, and the bowel is divided between clamps (fig. 1). When the ileocecal region is involved, as in ulcerative colitis, it is wise to remove a small segment

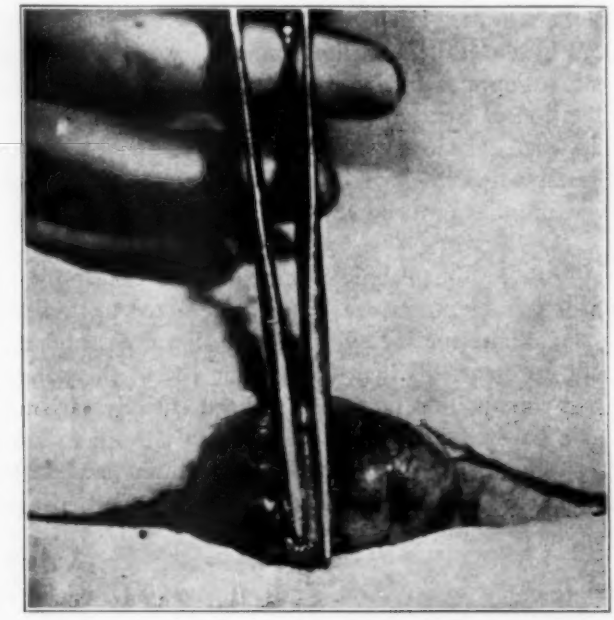

Fig. 1.—Ileum divided between clamps. In doing an ileostomy for ulcerative colitis, it is wise to remove a segment of ileum for immediate pathologic examination.

of ileum for immediate pathologic examination, as recommended by Dennis,[1d] to insure the area's being well above any pathologic process in the bowel. The distal stump of ileum is closed by oversewing the clamp, the suture line is reinforced by a second layer of interrupted fine cotton sutures and the closed stump is buried against the mesenteric leaf of the distal segment. The mesentery of the proximal segment is divided for a distance of 6 inches (15 cm.) at a point about 1½ inch (about 3.8 cm.) from the bowel margin, carefully preserving the distal vascular arcade (fig. 2). At this point, because of handling, exposure and edema, especially in greatly debilitated patients, the terminal 2

to 3 inches (5 to 7.6 cm.) of the ileal segment will show varying grades of cyanosis (fig. 2). Viability of the segment can be verified by administering 100 per cent oxygen to the patient for a few seconds with the face mask. The ileostomy is then brought out of the abdomen, and the proximal cut edge of mesentery is sutured to the peritoneum at the lower angle of the incision. Three seromuscular sutures of fine cotton fix the ileum at its antimesenteric and midlateral borders to the peritoneal edges of the lower half of the incision in such fashion that prolapse and rotation of the bowel will be prevented, and the ileum will arch downward as a spigot. The peritoneum is then closed with interrupted fine cotton sutures, and the anterior rectus sheath and skin are loosely

Fig. 2.—The ileal tube with divided mesentery is delivered from the abdomen. The distal color change may be noted.

closed around the ileal spigot, also with interrupted sutures of fine cotton (fig. 3). A thick split skin graft of suitable size is removed from the right thigh with the Blair-Brown knife and accurately applied to the exteriorized ileum. Accurate application of the graft under moderate tension is important. It is fixed to the abdominal skin edge at the base of the ileostomy by several interrupted sutures of fine cotton and so applied that the cut edges of graft meet on the antimesenteric aspect of the bowel, where they are united by a running cotton suture which does not include any of the bowel coats (fig. 4). At first, it was thought preferable to overlap the graft at the mesenteric border and to avoid suturing graft edge to graft edge. Experience with the remarkable

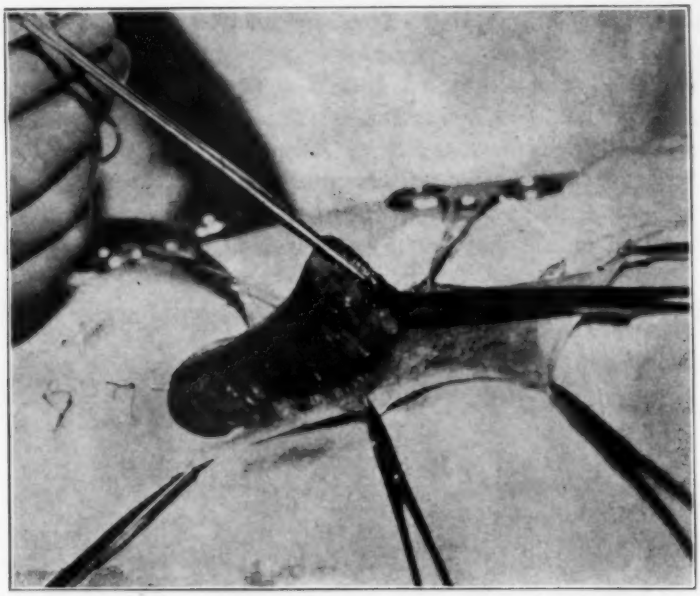

Fig. 3.—Abdominal incision closed loosely around the ileal tube. A skin graft is about to be applied.

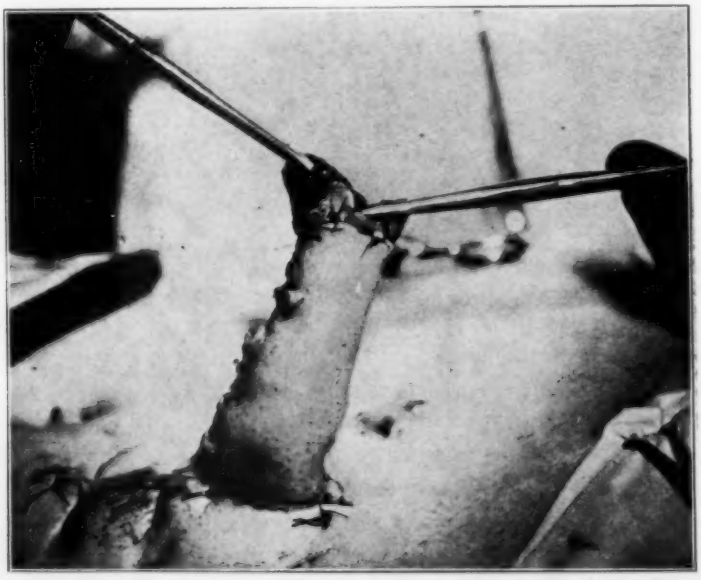

Fig. 4—Graft wrapped around the ileal tube and sutured in place, showing interrupted cotton sutures at base and tip; running cotton suture coapting graft edges.

excellence of the "take" of these grafts on the bowel wall, even in debilitated patients, and with the lumpiness produced along the mesenteric border by overlapping (fig. 6 and 8) has since indicated the safety and the desirability of accurately coapting the graft edges by suture along the antimesenteric border of the ileum (fig. 9). Vents in the graft are unnecessary and undesirable. A large Pezzar catheter is sutured into the stoma of the ileostomy and the grafted ileostomy carefully wrapped with a single layer of fine mesh petrolatum or bismuth tribromophenate ("xeroform") gauze. A voluminous dressing of machinist's waste is then applied, mainly to support the skin-covered ileostomy (fig. 5).

Fig. 5.—Pezzar catheter sewn in place. Voluminous supportive dressing of machinists' waste.

The usual postoperative measures are taken to preserve fluid, electrolyte and acid-base balance. The dressing is left undisturbed until the sixth or seventh postoperative day, at which time the sutures are removed from graft and incision and the Pezzar catheter is withdrawn. The somewhat ragged stoma left by the purse string suture should be neatly trimmed at this time to facilitate smooth mucocutaneous healing. An ordinary colostomy bag may be worn satisfactorily during the ensuing two weeks. At the end of this time the ileostomy will have assumed its permanent dimension and, if so desired, may be fitted with the custom-made device to be described later.

About three weeks postoperatively circumcision of the tip of the ileostomy, with an electrosurgical knife, may be done, either to shorten

an overly long spigot or to correct the stenosis which usually occurs at this time at the mucocutaneous junction of graft and bowel. At the present time circumcision is employed solely for the former purpose, because of the annoying and unsightly mucosal prolapse which results and because the stenosis, which is caused by fibroplasia in the submucosa and subserosa of the cut edge of the bowel wall, and not by graft contraction, will recur in due time (fig. 7).

Stenosis may be adequately controlled by periodic dilation with a large catheter or colon tube. Future experience may prove that a stenotic tip furnishes a desirable degree of control of the ileostomy discharge. One cooperative patient was satisfactorily managed for nearly two months, by evacuation of the partly obstructed ileostomy with a small tap water enema after each meal. There was little, if any, drainage

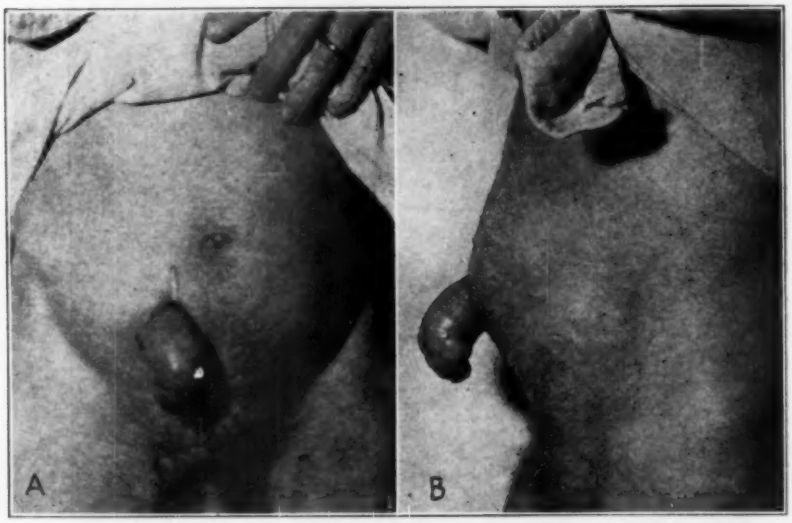

Fig. 6.—*A*, front view of completed ileal spigot three weeks later. Take of graft and absence of excoriation may be noted. *B*, side view showing spigot-like construction.

except at times of evacuation. In this case, however, distention of the ileostomy proximal to the stenotic tip produced a secondary valvelike obstruction at the level of the anterior rectus sheath, which caused uncomfortable cramping.

Fistula formation, which is an unfortunate, but not uncommon, complication in any type of ileostomy, has occurred in 2 cases in this series. In both instances the fistula developed on the antimesenteric surface of the external ileal spigot in locations where the bowel had been handled by instruments during the operative procedure (fig. 2). It has been assumed that intramural thrombosis and tissue necrosis caused by the operator's grasping the bowel wall with instruments was responsible.

Subsequently care has been taken to handle the bowel, at all times, solely with the well moistened gloved fingers. In both cases the development of the fistula was slow, premonitory signs of localized tissue death revealing themselves on the seventh postoperative day, but actual perforation being delayed between the second and third week postoperatively. Fecal drainage from these fistulas has been annoying but moderate in amount. Both fistulas healed spontaneously about two weeks after their formation.

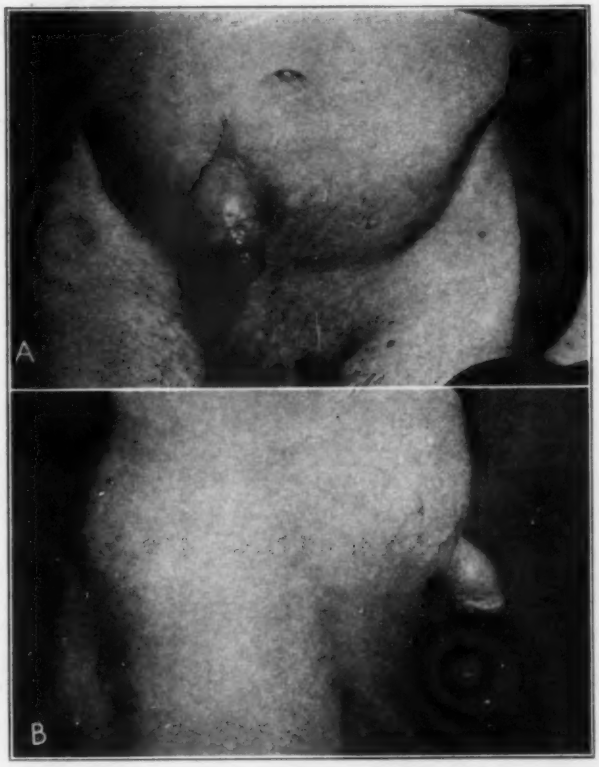

Fig. 7.—*A*, result of circumcision, showing undesirable mucosal herniation and recurrence of stenosing cicatrix at mucocutaneous junction. Absence of excoriation full four months after ileostomy, during which time patient had resumed his former occupation, may also be noted. *B*, side view.

COMMENT

The skin-covered ileostomy described can be fashioned with scarcely more trouble and time than is required for the construction of the more ordinary type described by Cattell and others. The average operating time has varied between sixty and ninety minutes, which is considerably less than the two and a half hour average period mentioned by Dennis for his painstaking and meticulous technic which produces simply a skin-buttressed nubbin on which to hang a Koenig-Rutzen bag.[1d]

Absence of painful skin excoriation is the greatest advantage of the Dragstedt ileostomy. The spigot-like construction facilitates direct transfer of the ileostomy discharge into a receptacle, avoiding leakage and spillage onto the surrounding abdominal skin, thus avoiding excoriation without the use of messy pastes and ointments.

Fig. 8.—*A*, photograph illustrating "lumpiness" produced by overlapping graft edges on mesenteric border. *B*, photograph showing the excellence of the take of these grafts, shown in this case by complete take of the "postage-stamp" graft near the tip of the spigot. This small graft was casually applied without adequate fixation at the time of the original operation to cover the defect left by the main graft of inadequate size.

The ease of fitting the penis-like spigot with a variety of receptacles, whether improvised, mass produced or custom made, is at once apparent. In 2 cases, pending the arrival of a proper bag, the patients were furnished with a number of surgeon's rubber gloves of large size, which were suspended around the waist by a length of umbilical tape. These makeshift appliances functioned satisfactorily for many weeks, completely preventing skin excoriation during this interval. Indeed, 1 patient, at the time of writing, had continued for eight months to prefer this lightweight simple makeshift appliance to the more cumbersome and elaborate appliances with which he had been furnished.

Fig. 9.—An extra long spigot, measuring nearly 5 inches (about 12.7 cm.) The flexibility may be noted.

During sleep, with the patient recumbent, back spillage from the nearly horizontal bag may be prevented by the application of simple cellulose cotton gaskets, which are easily made, as needed, by the patient. A longer bag for night time, which can lie to one side of the patient, may be helpful, or a Koenig-Rutzen bag may be used at night only, protecting the ileostomy skin covering with a cut-out condom or a thin layer of Ladd's paste.[3] Day time care is rather uncomplicated.

3. Ladd, W. E., and Gross, R. E.: Abdominal Surgery of Infancy and Childhood, Philadelphia, W. B. Saunders Company, 1941.

Certain instructions concerning dress should be followed, so that the ileostomy will not be injured by constricting corsets, girdles or belts. It is suggested that male patients wear suspenders for trouser support. Looser fitting male or female garments are, in general, advisable.

Although special custom-fitted bags are not necessary, a satisfactory appliance is specially made for this type of ileostomy by B. I. Shoelson of Chicago.[4] This embodies the principle of a hand-made silver base and sheath, curved to fit the ileostomy, to which can be attached by a screw-lock device a number of bags of different sizes for day and night wear. This type of appliance does have the advantage of protecting

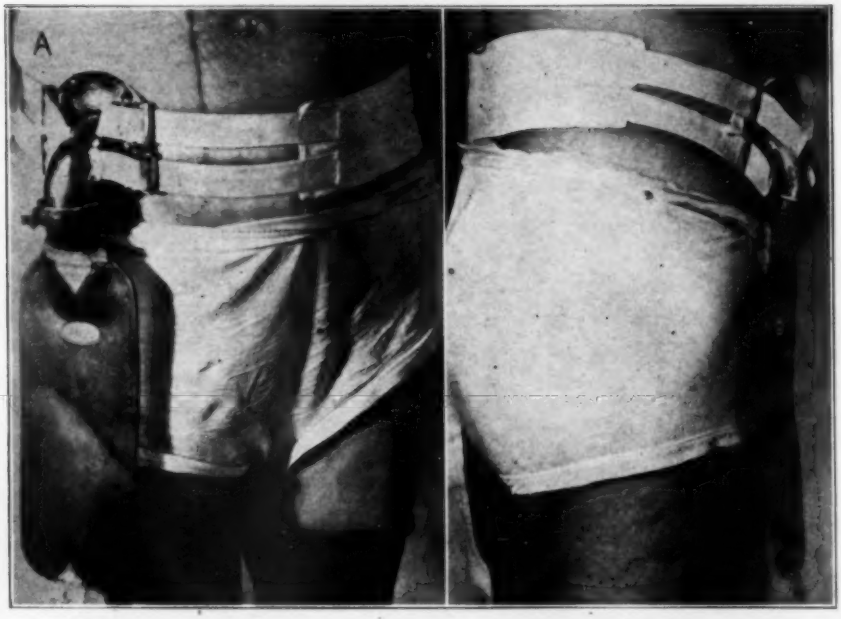

Fig. 10.—*A*, front view of Shoelson appliance. *B*, side view.

the ileal spigot from injury (fig. 10). The disadvantages of the weighty silver base should be overcome in the future by substitution of molded transparent plastic material, which also could be more easily cleaned.

CONCLUSIONS

1. The therapeutic efficacy of ileostomy is antagonized by the morale-destroying elaborateness and messiness of the dressing care needed to reduce or avoid skin excoriation in patients with ileostomy.

2. The Dragstedt skin-graft-covered ileostomy is the most satisfactory method of overcoming these shortcomings.

---

4. The Shoelson appliance may be obtained from Mr. B. I. Shoelson, 1448 East Sixty-Eighth Street, Chicago, Ill.

VALUE OF TRANSMETATARSAL AMPUTATIONS IN THE MANAGEMENT OF GANGRENE OF TOES

WESLEY FURSTE, M.D.
AND
LOUIS G. HERRMANN, M.D.
CINCINNATI

THE MANAGEMENT of gangrene of a toe or toes in patients with obliterative arterial disease, with or without diabetes mellitus, has gradually changed as a better understanding of the factors which are responsible for the spread of gangrene proximally in an affected foot has been acquired. The old axiom, "a gangrenous toe in a diabetic patient is always an indication for a supracondylar amputation," is no longer tenable. A natural, weight-bearing extremity can be obtained by a transmetatarsal amputation in many patients if proper precautions are exercised in the selection of cases and the execution of the operation.

We have employed this type of conservative amputation at the Cincinnati General Hospital since 1934, and we wish to present the facts which we believe are important in the selection of cases and the technical aspects of the operation which have given our best results.

In 1946, McKittrick [1] reported that between March 1944 and May 1, 1946, he and his associates had performed seventy-five transmetatarsal amputations. Eleven of these were considered to be failures, and 1 patient died of coronary heart disease after a higher amputation. In his report, he mentioned that in certain cases a small area of necrosis on the dorsal flap greatly prolonged the healing time. He stated:

> This experience, however, has been sufficient to cause the conviction that amputation at this level is practical, that with the use of penicillin it is safe, that the functional result is good and that it has been and will continue to be a method that—if carefully carried out following proper preparation and under the protection of penicillin in adequate dosage—will obviate amputation at a higher level in many cases.

MATERIAL

From May 1934 to Dec. 31, 1947, ninety-one transmetatarsal amputations were performed, eighty-five, or 93 per cent, of which have been carefully followed. Diabetic gangrene was the commonest condition for which the operation was

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

From the Department of Surgery of the College of Medicine, University of Cincinnati, and the Cincinnati General Hospital, Cincinnati.

1. McKittrick, L. S.: Recent Advances in the Care of the Surgical Complications of Diabetes Mellitus, New England J. Med. **235**:929, 1946.

carried out (table 1). For patients with diabetic gangrene, the average follow-up period if a higher amputation was not done was thirty-four months; with arteriosclerotic gangrene, nineteen months, and for frostbite gangrene, forty-five months. We considered these periods of follow-up study as adequate for our purpose. Patients who survived the operation and who did not have a higher amputation were not considered to have had an adequate follow-up period unless they were observed for a minimum of five months after operation. Many of these patients had previously had an amputation of one or more toes and an ulcer or sinus had developed which failed to heal (case 2).

In the selection of patients for amputation, age has not been a decisive factor (table 1). Diabetic patients as young as 45 years and as old as 84 years have submitted to amputation. An 84 year old white man with diabetic gangrene of his left fourth toe had an excellent result when he was examined twenty-two months after operation. The age range for arteriosclerotic patients has been 44 years to 80 years and for those with frostbite 38 years to 77 years.

TABLE 1.—*Age Range and Average Adequate Follow-Up Period of Patients Who Had Transmetatarsal Amputations*

Disease	Range of Age for 91 Amputations	Duration in Months of Average Follow-up for 85 Amputations	
		Higher Amputation Not Necessary	Higher Amputation Necessary*
Gangrene; diabetes	45-84	34	3.2
Gangrene; arteriosclerosis	44-80	19	5.2
Gangrene; frostbite	38-77	45	0.5
Gangrene; osteomyelitis	50-62	39	48
Gangrene; arterial embolism	68	29	....
Indolent ulcer, neurotrophic	14-33	37	8
Gangrene; thromboangitis obliterans	33	..	0.5

* The period was considered to extend from the time of the transmetatarsal amputation to the date of the higher amputation.

In patients with diabetes and arteriosclerosis, the dorsalis pedis and posterior tibial pulses were usually weak or absent; in those suffering from frostbite, the pulses were of fair to good quality in most instances.

Oscillometric studies were done in most of these cases, recording the total vascular pulsatility at the level to which the cuff was applied. These studies did not, however, distinguish a normal from a purely collateral circulation.

Infection of mixed character was present in the majority of cases. Osteomyelitis was not infrequently present and was often diagnosed preoperatively because of osseous changes in the roentgenograms.

METHOD

If there is evidence of cellulitis in addition to gangrene, an effort is made to overcome or localize it with meticulous care of the feet and chemotherapy, particularly with penicillin. In many cases, the associated infection had been controlled, but often only after a considerable period of time. Since penicillin has become easily available, usually 20,000 units have been given every three hours both before and after operation, but doses as small as 10,000 and as great as 50,000 units have been used. In 12 patients who received this antibiotic substance, the minimum total dose was 580,000 units and the maximum was 8,725,000 units. Of the 12 patients who received penicillin, 7 were diabetic, 3 were arteriosclerotic, 1 had frostbite gangrene and 1 had a neutrotrophic ulcer.

Incision through cellulitis while it is being made inactive by penicillin may lead to extension of the cellulitis, lymphangitis, tenosynovitis and suppurative arthritis. If, however, the infectious process is spreading rapidly in spite of therapy, amputation should be resorted to without further delay.

In cases of frostbite gangrene without spreading infection, a definite line of demarcation was allowed to develop and amputation was carried out at this level, the plan of treatment being similar to that followed by Shumacker [2] for gangrene in trench foot.

Recently, to aid the healing processes, ascorbic acid and one of the preparations containing the B vitamins have been used to supplement the diet.

Inhalation anesthetics were employed more frequently than spinal or local anesthetics. For the diabetic and arteriosclerotic patients, cyclopropane was the anesthetic agent of choice; but the combination of gas, oxygen and ether was used most frequently in cases of frostbite gangrene. Local anesthetics were not considered advisable because of the infection frequently associated with these cases. Spinal anesthetic was generally avoided because many of these patients had hypertension.

The operation was performed as follows: After routine preparation with soap, water, alcohol and ether, or with aqueous solution and tincture of ceepryn ("cetylpyridinium chloride") the distal half of the foot was draped. At first, a wire was firmly tightened about the foot distal to the proposed site of incision to lessen the possibility of extension of infection, but recently this has not been used. Since blood supply is poor in most of these patients, a proximal tourniquet is not employed. A transverse incision is made on the dorsum of the foot just proximal to the heads of the metatarsal bones (fig. 1) and extended around on the plantar surface. The incision is parallel to, but beyond a line through, the tarsometatarsal joints, so that the remaining segment of the fifth metatarsal bone is short. This segment, if too long, may become the base of an ulcer and be a source of pain. An attempt is made to place the plantar incision just about 1 cm. distal to the dorsal incision, so that the patient has a firm pad on the plantar portion of the stump and the operative scar is dorsal rather than plantar. The incision is developed, fine surgical gut being used to tie the vessels and to hold the tendons to the adjacent soft tissues. If the tendons are allowed to retract, they may carry bacteria with them and be responsible for a deep-seated infection. The soft tissues are held out of the way with moistened sponges. The periosteum is incised, and the metatarsals are divided in the middle third with a saw. Bone wax is used to control bleeding from the bone ends. Because of infection, the operative wound has not been closed, but has been dressed immediately with a saline compress, petrolatum gauze or fine mesh gauze. Recently in 1 case (case 4) under close postoperative scrutiny and with the use of penicillin, the stump was almost completely closed. The convalescent period was rather short, and the patient had an excellent functional result.

The initial dressing is applied a few days after operation. At first, saline or boric acid dressings are employed and, in event of gross infection, followed by dakinization (treatment with a buffered aqueous solution of sodium hypochlorite) or treatment with 3 per cent acetic acid. As the wound heals, either of the latter two dressings is replaced with foot soaks and wool fat gauze or fine mesh gauze, the former type of gauze being used if the skin appears hard and the latter if it is soft.

---

2. Shumacker, H. B., Jr.: The Surgical Treatment of Gangrene in Trench Foot, Surg., Gynec. & Obst. **83**:513, 1946.

If the stump becomes a red granulating area, pinch grafts, postage stamp grafts, or split thickness skin grafts are applied, the last of these three being our preference. Occasionally a local plastic procedure involving rongeuring away a small portion of bone or removal of a sequestrum (case 3) has been necessary to close a small ulcer of the stump.

In the postoperative period, the patient has been impressed with the importance of reasonable use of his stump, of avoidance of injury and infection, of hygienic measures, of the optimum level of the resting extremity and, if he is diabetic, of careful control of his diabetes.

Prostheses, such as a cotton pad, have been placed in the distal end of the shoe to prevent the foot from sliding forward. With such support, most patients have had little difficulty in standing or walking.

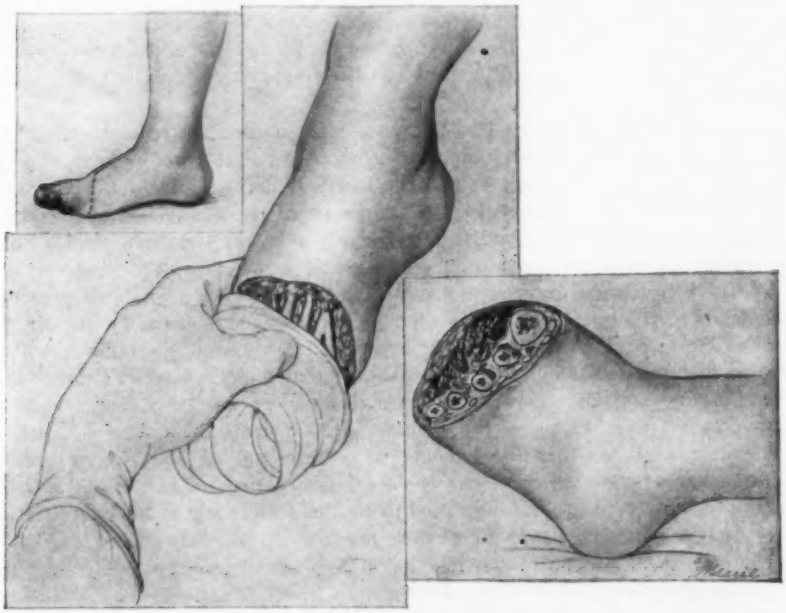

Fig. 1.—Technic of the transmetatarsal amputation, showing that the dorsal incision is slightly proximal to the plantar incision. It is important that the incision be parallel to, but of course beyond, a line through the tarsometatarsal joint, so that the remaining segment of the fifth metatarsal bone is short.

RESULTS

The criteria for evaluation of the results have been as follows:

Excellent: The stump remained healed permanently.

Good: The stump remained healed most of the time.

Fair: The stump not completely healed. Higher amputation one year or more later.

Poor: Higher amputation necessary within one year.

Questionable evaluation: The patient died from disease other than gangrene of lower extremities or suffered an injury of the extremities which was distinct from the original cause of the gangrene.

As table 2 indicates, the best results were obtained in cases of frostbite gangrene. For this condition, 67 per cent of the amputations healed permanently and 25 per cent remained healed most of the time, giving a total of 92 per cent. The 1 patient with a poor result was a 77 year old white man with pronounced arteriosclerosis who required a supracondylar amputation because of extending and infected gangrene. In 37 per cent of the cases of amputation for diabetes there was either an excellent or a good result and in 31 per cent of the cases of amputation for arteriosclerotic gangrene there were either excellent or good results. A report of typical cases follows:

TABLE 2.—*Results with Transmetatarsal Amputations*

Disease	Results for Amputations with Adequate Follow-Up						Number of Amputations Without Adequate Follow-Up
	Excellent	Good	Fair	Poor	Questionable Evaluation	Total	
Gangrene; diabetes	9	6	4	21	1*	41	1
Gangrene; arteriosclerosis	7	1	1	12	5†	26	3
Gangrene; frostbite	8	3	0	1	0	12	1
Gangrene; osteomyelitis	1	0	1	0	0	2	1
Gangrene; arterial embolism	1	0	0	0	0	1	0
Indolent ulcer, neurotrophic	0	0	1	1	0	2	0
Gangrene; thromboangiitis obliterans	0	0	0	1	0	1	0
Total	..	..	..	..	..	85	6

* The 57 year old diabetic white woman considered to have a questionable result died eight days postoperatively of a myocardial infarct demonstrated at necropsy.

† The patients with arteriosclerosis considered to have questionable results were as follows:

1. A 74 year old white man with an ulcer of his stump who died seven months after the transmetatarsal amputation because of probable carcinoma of the stomach.
2. A 58 year old white man who died one month after operation without definite determination of cause of death.
3. A 58 year old Negro with a well healed stump for six and a quarter months postoperatively, who suffered frostbite and required a low leg amputation.
4. A 68 year old white man who died one month postoperatively with bronchopneumonia.
5. A 67 year old white man who died two weeks postoperatively with pneumonia.

REPORT OF CASES

CASE 1.—E. S., a 59 year old white housewife with gangrene associated with diabetes mellitus (fig. 2), was admitted to the surgical service on March 13, 1941, because of an infection of her left foot. Three months prior to admission, there had developed a superficial ulceration over a bunion on the left foot. When the patient was seen by her family physician, he informed her that she had diabetes mellitus. While under his care, she blistered the medial side of her left great toe with foot soaks which were too hot, and a larger ulceration with spreading infection developed. On admission she appeared to be a well developed well nourished white woman who looked slightly younger than 59. Her temperature was 100.4 F., pulse rate 100, respiration 24 and blood pressure 160 systolic and 80 diastolic. Observations at physical examination were as follows: There were small bilateral varicose veins. The dorsalis pedis and posterior tibial pulses were not felt in either foot. Both feet were moderately cool, with the left slightly warmer than the right. There was a moderate swelling of the left foot and erythema extending from the great toe for 8 cm. proximally. There was a bunion with an infected ulcerated callus overlying it, the callus excreting pus from under

its edges. On the medial aspect of the great toe was an area 3 by 1 cm. of deep purple, representing an old second degree burn. The results of the remainder of the examination were not remarkable.

The urine in the initial test revealed sugar (3 plus) and contained no acetone. Laboratory data revealed: white blood cells 12,250, hemoglobin 12.6 Gm. per hundred cubic centimeters, red blood cells 4,580,000 and blood sugar 215 mg. per hundred cubic centimeters. Anteroposterior and oblique view roentgenograms of the left foot showed definite bone destruction in the distal portion of the first

Fig. 2 (case 1).—*A* and *B* show the foot just prior to transmetatarsal amputation on March 25, 1941. *C* was taken a few days before the application of small deep pinch grafts on April 29. In *G* (roentgenogram before operation) the bone destruction in the distal portion of the first metatarsal and of the two sesamoids over the head of the first metatarsal can be seen. In *H* there is no evidence of spur formation at the ends of the divided metatarsal bones.

metatarsal and in the proximal phalanx of the great toe. There also appeared bony destruction of the sesamoid bones over the head of the first metatarsal.

The patient was given routine foot care, and her diabetes, under the care of the diabetic service, was controlled with 0 to 30 units of crystalline insulin per day.

The infection appeared to be controlled but could not be overcome. On March 25, an open transmetatarsal amputation was performed with spinal anesthesia. With saline and then dakinized dressing, a granulating surface developed in the wound, and, on April 29, small deep pinch grafts were applied. The wound epithelialized, and the patient was discharged on June 5. By July 10 she was walking with crutches.

In December 1941 and January 1942, she was admitted for a subungual abscess of her right great toe and osteomyelitis of the distal phalanx of that toe. After removal of the toe nail and a small sequestrum from the distal phalanx, the toe healed.

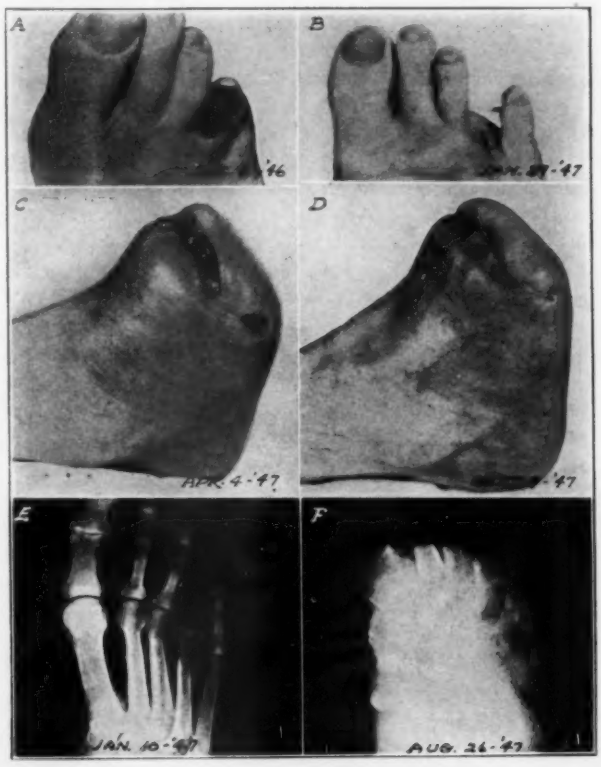

Fig. 3 (case 2).—In *D*, the typical dorsal scar is seen.

In *E*, the osteomyelitis of the head of the fourth metatarsal can be seen. In *F*, there is no evidence of spur formation of the divided ends of the metatarsals, and the remaining segment of the fifth metatarsal is not too long.

In 1942, 1945, 1946 and 1947, while she was wearing improper shoes, a very small ulcer of the stump developed, but in each case the ulcer healed. When last seen, on Feb. 3, 1948, the stump was healed. In view of the occasional small ulcerations of her stump, the patient is considered to have a good result.

Case 2.—O. S., a 73 year old white man with gangrene associated with diabetes mellitus (fig. 3), was admitted to the surgical service on Nov. 13, 1946, because of an infected right fourth toe. For five years he had had diabetes mellitus, which had been controlled by dietary regimen.

Six weeks prior to admission, a low grade infection of his right fourth toe had developed. There had been mild pain in this digit almost constantly, and there had been a moderate amount of exudate. The past history was not remarkable.

On physical examination he appeared to be a well developed and moderately obese conscious white man who appeared his stated age. His temperature was 98 F., pulse rate 84, respiration 22 and blood pressure 160 systolic and 94 diastolic. There was considerable reddening of the entire right fourth toe, with a light blackish discoloration of the dorsum and a small area of ulceration on the lateral aspect. The toe was rather tender. There was a moderate amount of redness of the adjoining toes. The foot was warm, and the dorsalis pedis and posterior tibial pulses were not palpable. Oscillometric studies revealed no pulsations in either lower leg. The results of remainder of the physical examination were not significant.

There was sugar (2 plus) and a questionable trace of acetone in the urine. Blood sugar was 166 mg. per hundred cubic centimeters, and carbon dioxide was 52 volumes per cent.

The patient was given routine foot care. Throughout his hospital stay, the diabetes was easily controlled with 10 to 20 units of regular or protamine zinc insulin daily and a diabetic diet.

The extent of the gangrene of the toe increased; hence, on December 9, the toe was amputated. The open wound failed to heal; on Jan. 10, 1947, roentgenograms revealed osteomyelitis of the head of the fourth metatarsal and demineralization of the foot bones. On January 29, therefore, an open transmetatarsal amputation was performed under cyclopropane anesthesia. Pathologic examination of the amputated portion of the foot revealed gangrene, arteriosclerosis and acute and chronic osteomyelitis.

On February 8, the wound was inspected for the first time and was covered with a slight amount of greenish exudate from which hemolytic Staphylococcus aureaus was grown. During the postoperative period the wound was treated locally with buffered aqueous solution of sodium hypochloride, 1 per cent acetic acid and finally soapy foot soaks; the patient was given penicillin, ascorbic acid and brewer's yeast. Gradually, the wound became clean and began to close.

On March 9, he was discharged. He continued the daily foot soaks at home and soon used only wool fat dressings. On August 15, he began to bear weight on the foot and soon began to walk. For support and protection he stuffed cotton in the toe of his shoe. When last seen, on Jan. 21, 1948, his right foot was in excellent condition, without any evidence of ulceration or drainage. This patient is considered to be an excellent result.

CASE 3.—M. T., a 60 year old white man with gangrene associated with diabetes mellitus (fig. 4), was admitted to the surgical service on June 9, 1946, because of an ulcer of his great toe. The lesion had developed three months previously when he had worn shoes which were too tight. He first became aware of his diabetes at a previous admission, in April 1946.

On physical examination, he appeared to be a fairly well developed and nourished conscious white man not acutely ill, who appeared his stated age. His temperature was 99.4 F., pulse rate 104, respirations 28 and blood pressure 150 systolic and 95 diastolic. The right great toe was slightly swollen and reddened. There was an ulcer 1.5 cm. in diameter with a necrotic base on the medial surface over the proximal interphalangeal joint. Motion in all joints was free and not painful. The pulses were considered to be satisfactory.

Urine contained a trace of sugar and no acetone. Fasting blood sugar was 185 mg. per hundred cubic centimeters, and the carbon dioxide was 64 volumes per

cent. Anteroposterior and oblique view roentgenograms of the right toes revealed arteriosclerosis of the vessels of the foot, with ulceration of the great toe.

On June 13 an open amputation of the right great toe was performed with cyclopropane anesthesia. Healing did not ensue; on July 5, an open transmetatarsal amputation was carried out. Some osteomyelitis of the second metatarsal appeared to delay healing. On August 9, therefore, the protruding bone was removed with a rongeur, and a split thickness graft was placed over the stump. The wound appeared to be healing satisfactorily, and on August 29 he was discharged from the hospital.

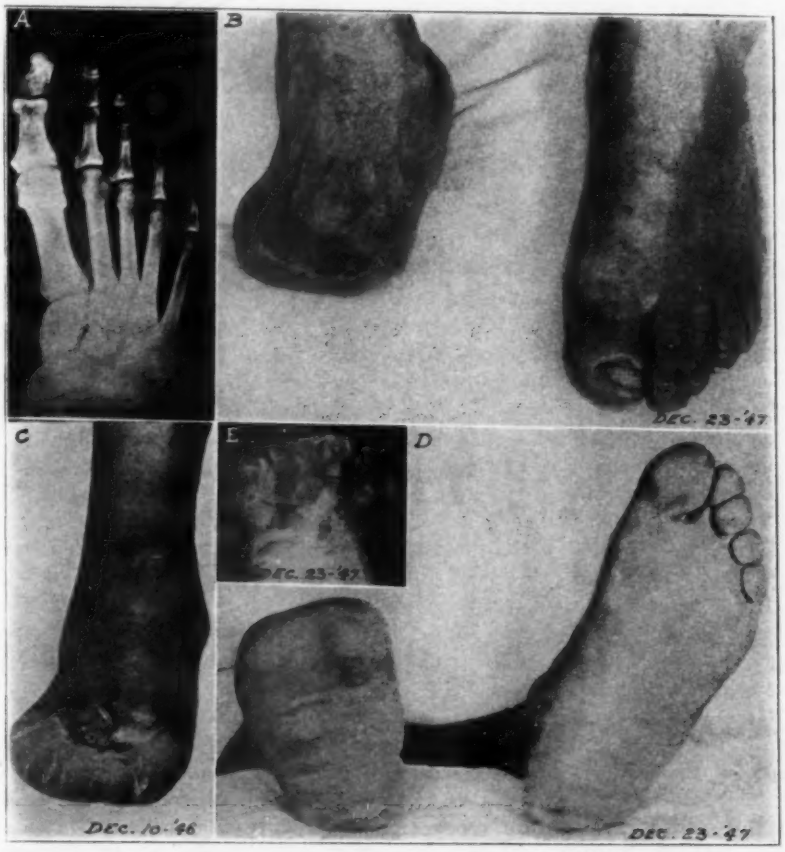

Fig. 4 (case 3).—In *A*, which was taken about three and a half weeks prior to the transmetatarsal amputation, the demineralization of the medial aspect of the distal phalanx of the great toe which was the site of the gangrene may be noted. *C* shows the stump on Dec. 10, 1946, when there was slight discharge from the area which necessitated the final plastic revision.

During his hospitalization he received 2,750,000 units of penicillin before and after the transmetatarsal amputation, the dosage being 15,000 or 20,00 units every three hours. At this and subsequent admissions the diabetes was controlled with small amounts of insulin.

While the patient was being followed in the clinic, the graft died. He was readmitted, and on October 22 a split graft was again applied. It also failed, and

on November 10 he was discharged with a granulating stump. No penicillin was given during this hospital stay.

On November 27 he was again admitted. With moist dressings the stump completely epithelialized, and, on Jan. 6, 1947, the stump was revised by removing more bone and closing the skin edges with interrupted silk sutures. No penicillin was given.

On Feb. 5, 1948, when the patient was last seen the stump was in excellent condition and enabled him to get about easily.

During December 1947, an ulcer developed on the medial aspect of the left great toe. Within three weeks the foot became cyanotic and cold, and on Jan. 2, 1948, a left low leg amputation was performed. In view of this last amputation, it was fortunate that a conservative course was followed in the management of the gangrene of the right great toe. It is particularly interesting that the right stump remained well healed in spite of the fact that one and a half years after the right metatarsal amputation the left foot became deprived of its blood supply and a high amputation was necessary. The result in this case is considered good.

CASE 4.—E. B., a white woman aged 47, with gangrene associated with diabetes mellitus (fig. 5), was admitted to the surgical service on Aug. 21, 1947, because of swelling and redness of her left toes.

The patient had been diabetic for about fifteen years. On July 13, 1946, she had had an amputation of her left second toe for diabetic gangrene and incision and drainage of a deep plantar abscess. While being followed in the clinic during the summer of 1947, an interdigital abscess developed between her left third and fourth toes, and because of an extension of the process she was admitted to the hospital.

On physical examination she appeared to be a well developed and well nourished conscious white woman who appeared her stated age. Her temperature was 99.8 F., pulse rate 104, respiration 24 and blood pressure 180 systolic and 95 diastolic. The left toes were greatly swollen. There was a yellowish purulent exudate from the third left toe. The left dorsalis pedis pulse was palpable and the left posterior tibial was not.

Urine contained sugar (2 plus) and no acetone. Pus from the foot was found to contain a coagulase positive nonhemolytic Staphylococcus aureus. Anteroposterior and oblique view roentgenograms revealed bone destruction in the metatarsals and phalanges of the left foot.

The foot was treated locally with moist compresses, and the diabetes was easily controlled with small doses of insulin and a diabetic diet. On August 30, a transmetatatarsal amputation was performed with cyclopropane anesthesia, and the skin edges were approximated very loosely with interrupted silk sutures. Before and after the operation, the patient received 2,320,000 units of penicillin. The wound healed slowly but steadily, without evidences of abscess formation. On September 18, the patient was discharged from the hospital.

After that, while being followed in the clinic, she made excellent progress. By the middle of October 1947 she was bearing weight on her foot. Cotton was used to fill the front of her shoe. On Feb. 9, 1948, the wound was well healed and the patient walked without difficulty. This patient is considered to have had an excellent result.

CASE 5.—C. P., a 56 year old Negro, with gangrene associated with arteriosclerosis, was admitted to the surgical service on Aug. 9, 1937, because of swelling and pain of his right great toe for two months. During this period, the toe had ulcerated and discharged large quantities of purulent material.

On physical examination the patient appeared to be a 56 year old Negro in fairly good general condition. His temperature was 100 F., pulse rate 112, respiration 22 and blood pressure 130 systolic and 60 diastolic. There was dry gangrene of the right great toe, with moderate edema and tenderness of the foot. No pulsations were felt in either foot. Oscillometric readings showed only very small movements for the lower legs.

On August 14, an open transmetatarsal amputation was performed with ether anesthesia. The wound became clean, and on September 3 split thickness postage

Fig. 5 (case 4).—On Oct. 21, 1947, the stump appeared as is shown and on Feb. 9, 1948, was in excellent condition. The slight vertical concavity on the stump is the result of an incision and drainage of a plantar abscess on July 13, 1946. The long plantar flap is considered to be an important part of a good stump.

stamp grafts were applied. Healing continued satisfactorily, and the patient was discharged on September 22. His stump slowly but permanently became epithelized.

The patient was readmitted in August 1940 because of the sudden onset of hemiplegia on the right side. Necropsy revealed cerebral arteriosclerosis with old and recent areas of softening and hemorrhage in the brain as well as cerebral edema and congestion. The stump of the right foot was covered with firm well healed scar tissue. In this case the result is considered to have been excellent.

CASE 6.—L. K., a 61 year old white man, with gangrene associated with arteriosclerosis (fig. 6), was admitted on the surgical service on June 6, 1945, because of a black left fourth toe.

Three weeks prior to admission a small lesion had developed on his left fourth toe, and, a few days later, the entire digit became gangrenous. Two weeks prior to admission, he noticed a small ulcer on the medial aspect of his left great toe.

On physical examination he appeared to be a well developed and well nourished white man, not acutely ill. His temperature was 98.6 F., pulse rate 96, respiration 20 and blood pressure 160 systolic and 110 diastolic. The left fourth toe appeared

Fig. 6 (case 6).—The stump was in excellent condition two years after the amputation. Because of pus-filled sinuses about several flexor and extensor tendons, incision and drainage had to be performed on Dec. 24, 1945, and resulted in the longitudinal scars seen in the photographs, which were taken on Sept. 29, 1947.

black and lifeless and was ulcerated in the region of the proximal interphalangeal joint. There was a crusted ulcer covered with exudate on the medial aspect of the left great toe.

On June 21 the fourth toe was removed without anesthesia. The gangrene, nevertheless, extended to the third and fifth toes. On September 11, roentgenograms revealed diffuse osteomyelitis of the distal end of the foot; hence, on September 18, an open transmetatarsal amputation was performed under cyclopropane anesthesia. The stump became grossly infected and, in spite of moist dressings, sinuses which

became filled with pus formed about several flexor and extensor tendons. On December 24, incision and drainage was performed with "penthothal sodium" (sodium 5-ethyl-5-[1-methylbutyl] thiobarbiturate) anesthesia. The wound began to granulate, and on Feb. 9, 1946, small deep grafts were successfully applied. Neither penicillin nor sulfadiazine was given to the patient. After discharge, on February 14, the stump epithelized completely and was well healed on Sept. 30, 1947, when the patient was last seen. This is considered to be an excellent result.

CASE 7.—J. B., a 55 year old Negro, with gangrene associated with frostbite, was admitted on the surgical service on Jan. 25, 1940, because of frostbite of both feet.

On the evening prior to admission, the patient suffered frostbite of both feet when he fell asleep beside an outdoor fire. When he awoke in the morning, both feet were numb and cold. After he warmed them beside a fire, they became blistered.

On physical examination, he appeared to be a poorly nourished conscious Negro, who was extremely dirty. His temperature was 97 F., pulse rate 92, respiration 24 and blood pressure 130 systolic and 70 diastolic. There were definite blebs on the dorsum of all the right toes and the left great toe. The right toes were cool and lacked sensation; the left toes were warm and had sensation. There was slight pedal edema of both feet and ankles. The pulses were palpable on both feet.

A line of demarcation developed on the right foot just proximal to the metatarsal-phalangeal joint; on January 31, a right open transmetatarsal amputation was performed with cyclopropane anesthesia. By February 13, there was a good granulating wound, and split thickness postage stamp grafts were applied to the right stump. He was not given penicillin or sulfadiazine. On May 14, nine days before discharge from the hospital, two small metatarsal spurs were removed.

In September and December the patient had slight drainage from the right stump. During the eight year follow-up period the foot remained healed, and it has enabled him to walk almost normally. This is considered to be an excellent result.

ANALYSIS

Reasons for Failure.—The main causes of failure have been extending gangrene, in most cases resulting from an inadequate blood supply, extending cellulitis associated with lymphangitis, persistent osteomyelitis, tenosynovitis, proximal extension of the infection with development of suppurative tarsal arthritis, generalized debility of the patient, or a combination of one or more of these factors producing a nonhealing ulcer of the stump. The point where the operative incision is most likely to break down is on the dorsum of the foot in the center. The explanation for this site of breakdown is not clear, but successful healing of this area will overcome one of the present major difficulties.

Penicillin.—Penicillin appeared to aid in control of the local invasiveness of the infection. In 6 of the 12 cases in which it was given, its administration was associated with fall of an elevated temperature to a normal level.

Indications and Contraindications.—The transmetatarsal amputation is justifiable, but it is to be used only in carefully selected cases. Injudicious choice of patients, particularly those with pronounced vascular insufficiency of the leg and foot, is likely to be followed by a large percentage of higher amputations and hence a poor result. In many instances, the operation can be offered only with the distinct under-

standing and a clear explanation of the fact that with the impaired blood supply success is not certain and that a higher amputation may be necessary.

In deciding which patients are suitable for operation, a careful assessment of the circulatory supply and of the amount and rate of extension of infection is fundamental. This amputation is indicated when the following conditions are present:

1. Gangrene of one or more toes with the gangrene spreading to the region of the metatarsal-phalangeal joints, so that healing will probably not occur after the amputation of a toe or toes.

2. Gangrene as noted under (1) but without spreading cellulitis of the foot.

3. Persistent ulcer following amputation of a toe or toes.

4. Fair circulatory balance.

5. Necessity for weight-bearing stump.

This amputation is not indicated under the following conditions:

1. Gangrene of most of the toes, with the gangrene extending over the metatarsals.

2. Gangrene of one or more toes, with spreading cellulitis of the foot.

3. High grade arterial insufficiency.

Whether or not the patient is the wage earner is of some importance in making the decision for either a transmetatarsal or a higher amputation. If it is essential for the patient to lose as little time as possible from work and if his hospital stay must be made as short as possible because of limited finances, the supracondylar amputation may be recommended as affording a good stump in a relatively short time. It will, however, deprive the patient of a natural weight-bearing lower extremity.

Inasmuch as results are uniformly excellent or good with the transmetatarsal amputation for frostbite gangrene, its use for this condition is strongly recommended when a line of demarcation has developed and indicates that none of the toes can be saved.

Each case must be considered by itself. No single rule can be stated which adequately and completely defines when the transmetatarsal amputation is to be employed and when it will be successful.

Advantages.—The transmetatarsal amputation gives a natural weight-bearing lower extremity, which enables the patient to walk successfully and to carry on usual activities, provided that the blood supply is sufficient to enable healing of the operative wound and provided that neurotrophic anesthesia and associated susceptibility to pressure are not present. Such an amputation is very valuable to a person who already has had a supracondylar amputation of the other lower extremity or who, as a young or middle-aged man, needs a lower

extremity which has normal knee and ankle joints. Persons with this amputation do not have an equinus deformity because the muscle balance of the foot has been preserved by not disturbing the insertions of the tibialis anterior, the tibialis posterior and the peronei muscles and by attaching to the adjacent soft tissues the proximal ends of the divided long flexor and extensor muscles. There is normal movement of the ankle. The person's weight is borne on the bottom of the foot, and not on the stump.

When a long plantar flap has been obtained, with a resulting dorsal scar, the stump is not a source of pain and is not tender. Patients with supracondylar amputations not infrequently complain of phantom limb pain; those with transmetatarsal amputations only rarely state they have such pain in their amputated toes. This amputation is much less of a mental shock than the supracondylar; the person will endeavor to care for himself and will not be dependent on others. If the amputation is performed through the distal portion of the metatarsals, the possibility of suppurative arthritis of the tarsal joints is small.

TABLE 3.—*Hospital Deaths with the Transmetatarsal Amputation*

Disease	Total Number of Transmetatarsal Amputations	Number of Hospital Deaths	Mortality, Percentage
Gangrene; diabetes*	42	6	14
Gangrene; arteriosclerosis†	29	4	14
Gangrene; frostbite‡	13	1	8
Gangrene; osteomyelitis	3	0	0
Gangrene; arterial embolism	1	0	0
Indolent ulcer, neurotrophic	2	0	0
Gangrene; thromboangitis obliterans	1	0	0

* Causes of death among diabetic patients were bronchopneumonia (2), extending gangrene (2), myocardial infarct (1) and undetermined cause (1).

† Causes of death among patients with arteriosclerosis were bronchopneumonia (2), pneumonia (1) and undetermined cause (1).

‡ Cause of death in 1 case of frostbite in a 77 year old white man with pronounced arteriosclerosis was probably myocardial infarction.

The disadvantage of the transmetatarsal amputation is that operation may later be necessary at a higher level. With more accurate selection of cases, however, a smaller percentage of higher amputations should result.

Hospital Mortality Rate.—The hospital mortality rate for this series of ninety-one transmetatarsal amputations is given in table 3.

In 1945, Herrmann and Gibbs [3] reported a 22.7 per cent operative mortality rate for 110 cases of major amputation in arteriosclerosis obliterans with diabetes mellitus and 23.9 per cent for 88 cases of amputation in arteriosclerosis obliterans without diabetes mellitus. Combining

3. Herrmann, L. G., and Gibbs, E. W.: Phantom Limb Pain, Am. J. Surg. **67**:168, 1945.

4. Silbert, S.: Amputation Below the Knee for Gangrene in the Diabetic, Am. J. Digest. Dis. **11**:394, 1944.

the statistics of twelve representative metropolitan hospitals from 1916 to 1940, Silbert [4] found 300 deaths in 637 cases of thigh amputation in diabetic persons, a mortality rate of 47 per cent. McKittrick [1] reported 86 deaths in 609 cases from the New England Deaconess Hospital for the period 1923-1941, a percentage of 14.1, and 10 deaths in 212 cases from 1941-1945, a percentage of 4.7 for amputations through or above the middle part of the lower leg in diabetic patients.

In the present series of transmetatarsal amputations there has been a 14 per cent mortality rate for 42 cases of diabetic gangrene and 14 per cent for 29 cases of arteriosclerotic gangrene. Only 1 death occurred in the 13 patients with frostbite, a mortality rate of 8 per cent. The patient was a 77 year old white man who had severe arteriosclerosis. Since gangrene had developed after exposure to cold weather, it was believed that this case should be considered a case of frostbite rather than of arteriosclerotic gangrene.

SUMMARY AND CONCLUSIONS

During a fourteen year period, ninety-one transmetatarsal amputations have been performed at the Cincinnati General Hospital for gangrene of the toes, due most frequently to diabetes, arteriosclerosis and frostbite. The indications and contraindications for the operative procedure have been determined. There must be a judicious selection of cases; the operation must be carefully performed, and preoperative and postoperative care must be adequate. Seven typical cases are presented.

This amputation is safe, has a low mortality rate and is rarely associated with phantom pain. Ninety-two per cent of the patients with amputations for gangrene associated with frostbite, 37 per cent for gangrene associated with diabetes and 31 per cent for arteriosclerotic gangrene gave good functional lower extremities obviating a higher amputation.

With the insight gained from this fourteen year experience, with the help of the antibiotic agents and with a careful selection of cases, it is thought that a greater percentage of successful transmetatarsal amputations can be obtained.

DISCUSSION

DR. FRANCIS H. STRAUS, Chicago: In the Cook County Hospital of Chicago I have many times used the procedure described by Dr. Furste and Dr. Herrmann. I have used it principally for frostbite of the feet and only occasionally for ischemic gangrene. A long plantar flap and a short dorsal flap are made and left open. The wound is packed until the infection has subsided and necrotic fascia and tendons have disappeared. Then the long plantar flap can be brought to the margin of the dorsal flap. This is easily done with elastic rubberband traction. This saves a second operative procedure.

ROLE OF ANTIBIOTICS IN MASKING INFLAMMATORY PROCESSES

ANTHONY R. CURRERI, M.D.

ERWIN R. SCHMIDT, M.D.

AND

JOHN T. MENDENHALL, M.D.

MADISON, WIS.

ANTIBIOTICS unquestionably have played a major role in the brilliant surgical advances of the past decade. Extensive radical surgical treatment hitherto associated with a grave prognosis because of complicating infections is now performed in all large general hospitals because of the availability of these chemotherapeutic agents. It is also common knowledge that many infections, such as carbuncle, empyema, mediastinal abscess, acute cholecystitis and actinomycosis, respond so favorably to these drugs that in many instances surgical intervention is unnecessary.

Unfortunately, these dramatic and often awe-inspiring results have projected into the minds of many physicians a false sense of value regarding the powers of these drugs. Physicians employing these drugs as panaceas without due regard to surgical principles often are lulled into a feeling of security, and as a result of prolonged or injudicious use cause either masking of the true clinical picture or irreversible pathologic changes.

There has been a recent trend to treat infections with sulfonamide drugs and penicillin in order to preclude the possibility of an operation. Surgical services everywhere now see more and more cases in which physicians tried to control surgical infections by means of antibiotics with disastrous end results. The antibiotics are powerful adjuncts and should be used as such. They cannot replace established principles of rest, surgical drainage, parenteral fluids, heat and, even more important, good sound judgment.

Certainly many infections undergo resolution, provided that the causative agent has been corrected. In others, where the causative factor is still present, perforation occurs just as promptly because the antibiotics, if effective, will diminish the number of organisms; but edema, obliteration of vessels, necrosis and perforation of the wall will

---

From the Department of Surgery, University of Wisconsin Medical School, Madison, Wis.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

necessarily ensue. Inasmuch as the drugs have retarded the inflammatory reaction, nature walls off the peritonitis. If the perforation becomes sealed, the abscess in a certain percentage of cases will absorb. Every surgeon has observed patients exhibiting this response. The problem is different when the perforation persists, because the antibiotic action frequently will mask either the toxic picture or abdominal signs pointing to peritonitis.

The following 2 cases illustrate how the clinical picture is altered by administration of penicillin in acute abdominal infections.

ILLUSTRATIVE CASES

Case 1.—A 55 year old white woman had typical acute cholecystitis symptoms on the tenth day following nephrectomy. Conservative treatment, including par-

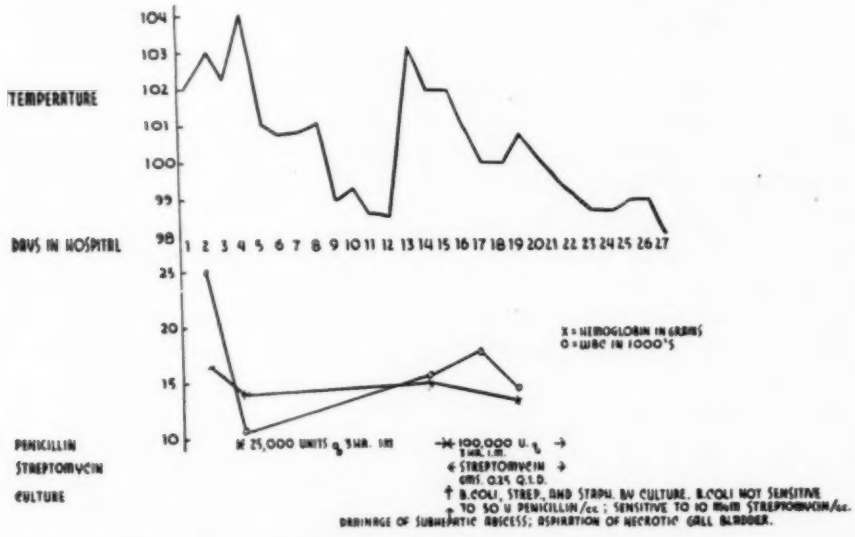
TEMPERATURE
104
103
102
101
100
99
98
DAYS IN HOSPITAL
1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23 24 25 26 27
25
20
15
10
X = HEMOGLOBIN IN GRAMS
O = WBC IN 1000'S
PENICILLIN
STREPTOMYCIN
CULTURE
@ 25,000 UNITS q 3 HR. IM
100,000 U. q 3 HR. I.M.
STREPTOMYCIN GMS. 0.25 Q.I.D.
B.COLI, STREP., AND STAPH. BY CULTURE. B.COLI NOT SENSITIVE TO 50 U PENICILLIN/cc; SENSITIVE TO 10 mgm STREPTOMYCIN/cc.
DRAINAGE OF SUBHEPATIC ABSCESS; ASPIRATION OF NECROTIC GALL BLADDER.

Fig. 1.—Graph of data in case 2, perforated gallbladder.

enteral use of penicillin, caused a remarkable subsidence in fever and toxicity, and at no time was abdominal muscle spasm or rigidity noted. However, in view of two recurring attacks of acute pain, mounting white blood cell count and sedimentation rate, the abdomen was explored fifty hours after the onset, and a large abscess with a perforation in the lower end of the gallbladder was discovered.

Case 2.—A 50 year old physician (fig. 1) was admitted to the Wisconsin General Hospital in shock, with extreme toxicity, cyanosis and abdominal distention. The history was suggestive of either acute cholecystitis or gastroenteritis. The immediate response to conservative treatment and penicillin was rather dramatic; the sole complaint was lack of appetite, and ambulation of the patient was permitted. On the tenth and eleventh day after admission, an icteric tint of the sclera, slight tenderness, a mass in the right upper quadrant of the abdomen and rising leukocyte count were observed. At operation there was a large subhepatic abscess, from which a necrotic gallbladder was aspirated.

Comment.—The recognized protective action of antibiotics against infections has led to their general use postoperatively, particularly when bacterial contamination may, or actually has, taken place. One must be mindful of the fact that in the event these agents are ineffective in controlling the infection the clinical picture may be confusing. Case histories will be presented which will show how these drugs produced departures in the classic clinical picture.

CASE 3.—A 53 year old man (fig. 2) had a recurring gastric carcinoma complicated by an obstruction at the site of the previous gastrojejunal anastomosis. Postoperatively, penicillin was given, and except for anorexia the progress seemed satisfactory. On the eleventh day postoperatively there were signs of abdominal distention and a high leukocyte count. Exploration revealed a large abscess, and in view of mixed organisms of Bacillus coli, staphylococci and streptococci, streptomycin

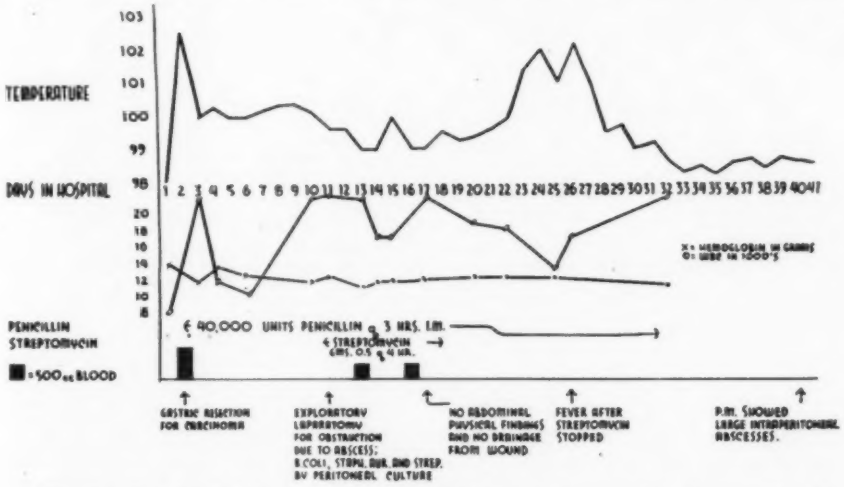
TEMPERATURE
DAYS IN HOSPITAL
PENICILLIN
STREPTOMYCIN
GASTRIC RESECTION FOR CARCINOMA
EXPLORATORY LAPAROTOMY FOR OBSTRUCTION DUE TO ABSCESS; B.COLI, STAPH. AUR. AND STREP. BY PERITONEAL CULTURE
NO ABDOMINAL PHYSICAL FINDINGS AND NO DRAINAGE FROM WOUND
FEVER AFTER STREPTOMYCIN STOPPED
P.M. SHOWED LARGE INTRAPERITONEAL ABSCESSES.

Fig. 2.—Graph of data in case 3, peritonitis secondary to gastric resection for recurrent carcinoma.

was also used. Within seven days the abdominal drainage and muscle rigidity and spasm disappeared. In spite of the paucity of signs the course was downhill, and necropsy showed large intraperitoneal abscesses.

CASE 4.—A 65 year old man (fig. 3) was admitted to the medical service for obstruction secondary to a Richter hernia. Under conservative management perforation of the small bowel took place, with repair approximately nine hours later. With streptomycin and penicillin therapy this patient, as the previous one, early postoperatively showed little toxicity and a low grade fever, which on occasions reached high peaks. Autopsy showed a large intraperitoneal abscess.

CASE 5.—A 26 year old girl (fig. 4) had a Finsterer type of gastric resection for actively bleeding duodenal ulcer with obstruction. Contrary to the previous cases this patient was rather toxic and had a septic fever, but the white blood cell count, except on two occasions, was always less than 12,000. Exploration on the twentieth and thirtieth days postoperatively failed to reveal abscesses. Autopsy showed extensive peritonitis with abscess formation only retroperitoneally.

Comment.—One can conclude that in the presence of antibiotic therapy the classic syndrome of shock, toxicity, leukocytosis, fever and boardlike muscular rigidity associated with peritonitis will not occur often. There are no constant pathognomonic symptoms, and one must

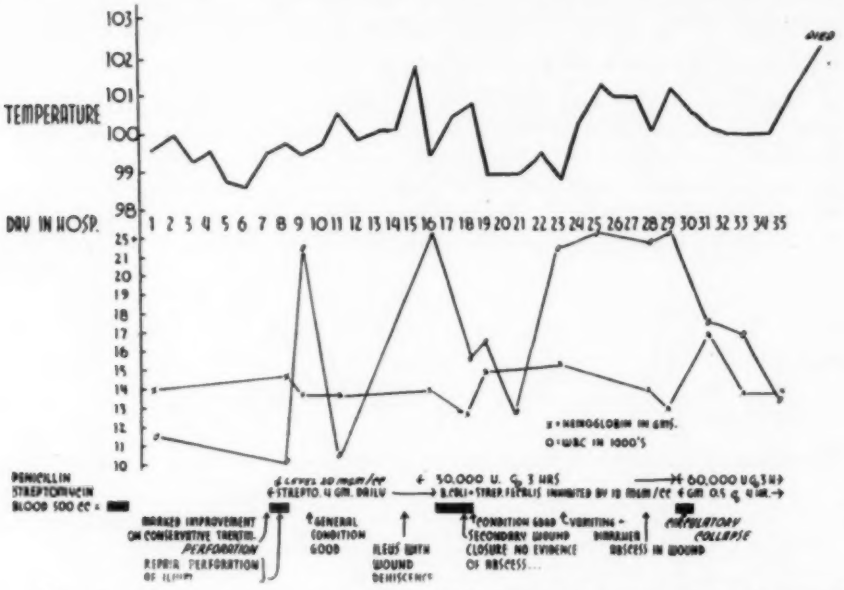

Fig. 3.—Graph of data in case 4, Richter's hernia with perforation and generalized peritonitis.

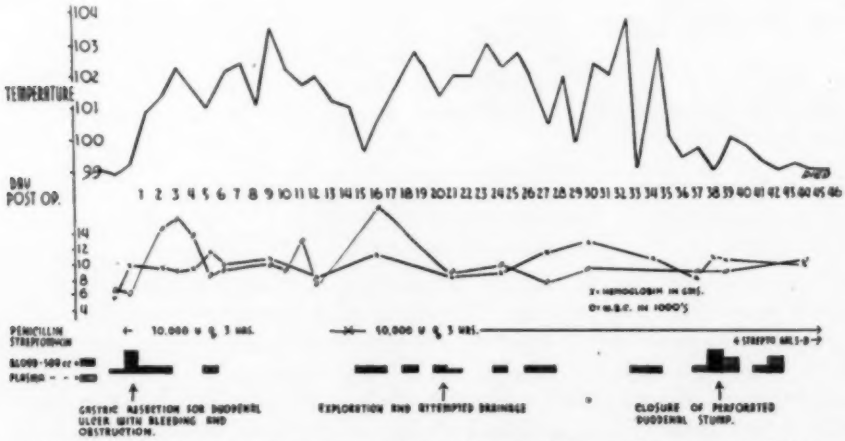

Fig. 4.—Graph of data in case 5, gastric resection, perforation of duodenal stump and extensive peritonitis with retroperitoneal abscess formation.

rely on the failure of the patient to respond properly as the first indication of imminent complications, such as peritonitis and abscess formation.

In acute thoracic infections the failure to appreciate the limitations of antibiotics often ends disastrously. Acute pulmonary abscesses usually heal with dependent postural drainage and penicillin. Many patients with empyema have been cured, without surgical intervention, by instillation of penicillin into the pleural space after aspiration of the pleural pus. At the same time, conservative therapy should not be continued until irrevocable changes take place in the thorax.

CASE 6.—A 55 year old man in whom localized empyema developed after pneumonia received penicillin parenterally for nine weeks because his physician could not find the localized empyema cavity. At the time of admission the pleura was so thick that after the pleural space was completely aspirated the lung did not expand at all. Pulmonary decortication was necessary in order to expand the lung and obliterate the empyema space.

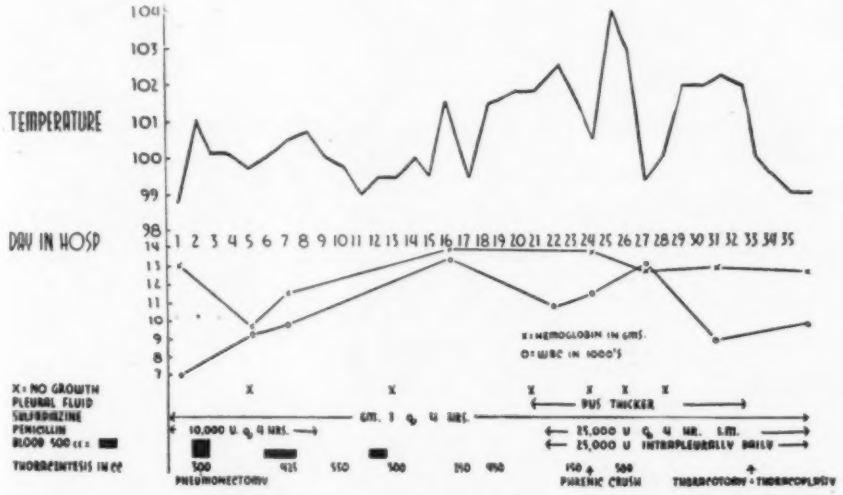

Fig. 5.—Graph of data in case 8, congenital cystic lung with postpneumonectomy empyema.

CASE 7.—A 16 year old high school athlete had streptococcic empyema. The lack of toxicity and very thin pus resulting from sulfamerazine therapy led us to procrastinate regarding drainage of the pleural space. It suddenly struck us that the roentgenograms demonstrated considerable pleural thickening. Fortunately, the lung expanded after rib resection.

Comment.—The aforementioned and the following cases point to the minimal toxicity and considerable delay in pus formation when antibiotics incompletely control the pleural infection. However, all these cases have in common the fact that there is reformation of pleural fluid despite repeated thoracenteses and instillation of antibiotics.

CASE 8.—T. G., a 22 year old girl (fig. 5) had a pneumonectomy performed for congenital cystic lung. Her general condition was satisfactory enough so that she

was ambulatory on the sixth postoperative day. There was rapid formation of pleural fluid, requiring repeated aspirations. Until the twenty-fourth day postoperatively the aspirated fluid showed no organisms.

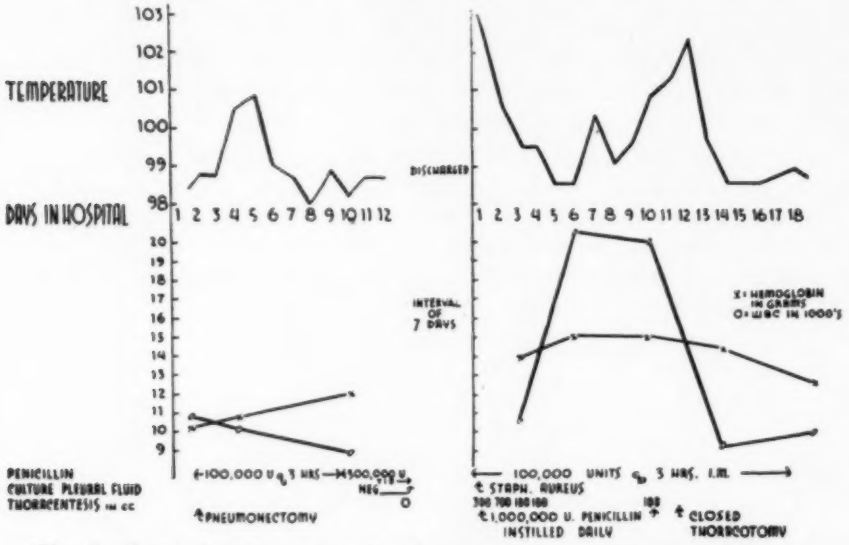

Fig. 6.—Graph of data in case 9, actinomycotic lung with pneumonectomy.

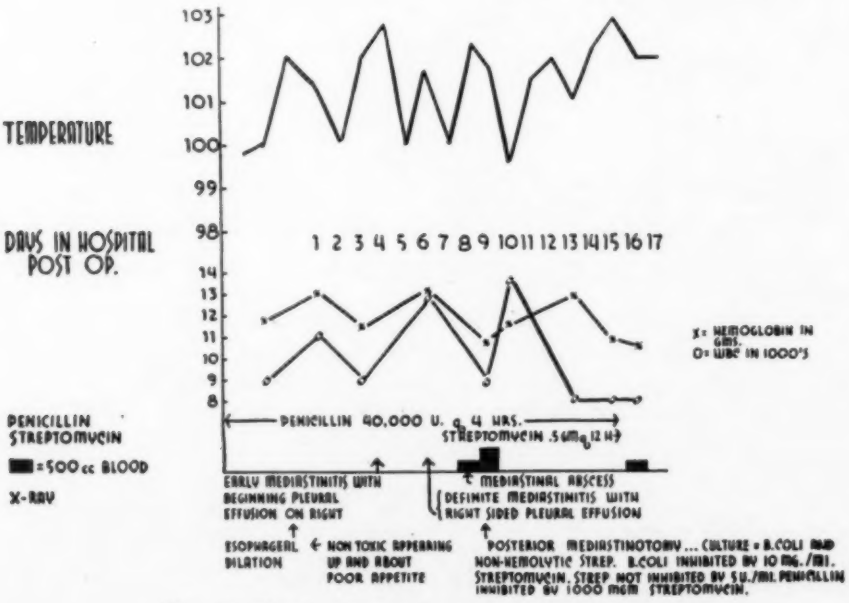
TEMPERATURE
DAYS IN HOSPITAL POST OP.
x = HEMOGLOBIN IN GMS.
O = WBC IN 1000'S
PENICILLIN
STREPTOMYCIN
= 500 cc BLOOD
X-RAY
PENICILLIN 40,000 U. q 4 HRS.
STREPTOMYCIN .5 GM q 12 H
MEDIASTINAL ABSCESS
EARLY MEDIASTINITIS WITH BEGINNING PLEURAL EFFUSION ON RIGHT
DEFINITE MEDIASTINITIS WITH RIGHT SIDED PLEURAL EFFUSION
ESOPHAGEAL DILATION
NON TOXIC APPEARING UP AND ABOUT POOR APPETITE
POSTERIOR MEDIASTINOTOMY ... CULTURE = B.COLI AND NON-HEMOLYTIC STREP. B.COLI INHIBITED BY 10 MG./ML. STREPTOMYCIN. STREP NOT INHIBITED BY 5 U./ML PENICILLIN INHIBITED BY 1000 mgm STREPTOMYCIN.

Fig. 7.—Graph of data in case 10, mediastinitis.

CASE 9.—B. L., a 24 year old girl (fig. 6) had a splendid convalescence following a pneumonectomy for an actinomycotic lung. She was discharged on the tenth postoperative day with no fever, leukocytosis or toxicity, and the pleural fluid

showed no organisms. Penicillin therapy was stopped on day of discharge. The patient was readmitted to the hospital seven days later in a toxic state and the pleural aspiration revealed Staphylococcus aureus.

CASE 10.—A patient (fig. 7) had mediastinitis resulting from perforation following esophageal dilatation. (The inhibitory reaction of the antibiotics was insufficient to control the infection, but clinically the patient was not toxic and was up and about.) Her only complaints were compression sensation in the chest, anorexia and, later, dyspnea due to pleural effusion. Because of this apparently good response to penicillin therapy, operation was not done until the abscess became extremely large and compressed the surrounding structures.

CONCLUSION

We should like to emphasize that while antibiotics are of inestimable value in the control and treatment of infection, they place on the surgeon a grave responsibility, for when complications develop, the classic clinical picture is masked, requiring, therefore, the exercising of the best possible judgment.

PREOPERATIVE PLACE OF STREPTOMYCIN IN OPERATIONS ON THE BOWEL

H. S. MORTON, M.B., F.R.C.S.
AND
F. SMITH, M.B., F.R.S.C.
MONTREAL, CANADA

IN ALL surgical procedures asepsis is essential, and in preparing for operation on the bowel it would seem desirable to render the lumen as clean as possible.

About 75 per cent of the flora of the normal adult colon consists of gram-negative organisms, mainly enterobacteriaceae, though there are decided differences in the various segments of the gut. The remainder are mainly streptococci, anaerobes and organisms which probably have not regularly been studied in culture. Occasionally exceptional numbers of Proteus, Pseudomonas and even Staphylococcus pyogenes may be present. These appear often to cause pronounced degrees of disfunction, although they are not commonly regarded as gut pathogens in the strict sense. There may, in addition, be true pathogens of the Salmonella or dysentery groups.

Until the introduction of streptomycin by Schatz, Bugie and Waksman [1] there had been no antibiotic acting on these gram-negative bacillary organisms. Because of the concentrating effect of renal excretion, streptomycin found early favor in the therapy of nontuberculous infection of the renal tract. Similarly recognized was its activity against Haemophilus influenzae, whether in the meninges or the respiratory tract, and on organisms such as Friedländer's bacillus. Its major importance in tuberculosis is a more recent and sedate development, as is the influence, to be described, on surgery of the colon.

The pharmacology of streptomycin resembles that of penicillin closely, and intramuscular injection is the chief method of administration. Indeed, it is not absorbed from the alimentary tract. Herein,

---

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

From Department of Surgery, Royal Victoria Hospital, Queen Mary Veterans' Hospital and McGill University (Dr. Morton), and the Department of Bacteriology, McGill University, Montreal, Canada (Dr. Smith).

1. Schatz, A.; Bugie, E., and Waksman, S. A.: Streptomycin: A Substance Exhibiting Antibiotic Activity Against Gram-Positive and Gram-Negative Bacteria, Proc. Soc. Exper. Biol. & Med. **55**:66, 1944.

however, together with its decided stability, lies one of its cardinal virtues from the point of view of the present paper.

We have demonstrated previously [2] that it is extremely useful topically in the peritoneal cavity, and now we wish to show its value when given orally to prepare the intestines preoperatively in such conditions as ulcerative colitis, carcinoma of the colon, tuberculosis of the intestines, transplantation of the ureter, abdominoperineal resections and particularly in any form of anastomosis of the bowel.

In 1946 we began using streptomycin both by mouth and by intramuscular injection in a series of ulcerative colitis cases, using 1 Gm. orally and 3 Gm. intramuscularly per day. The sterilizing effect

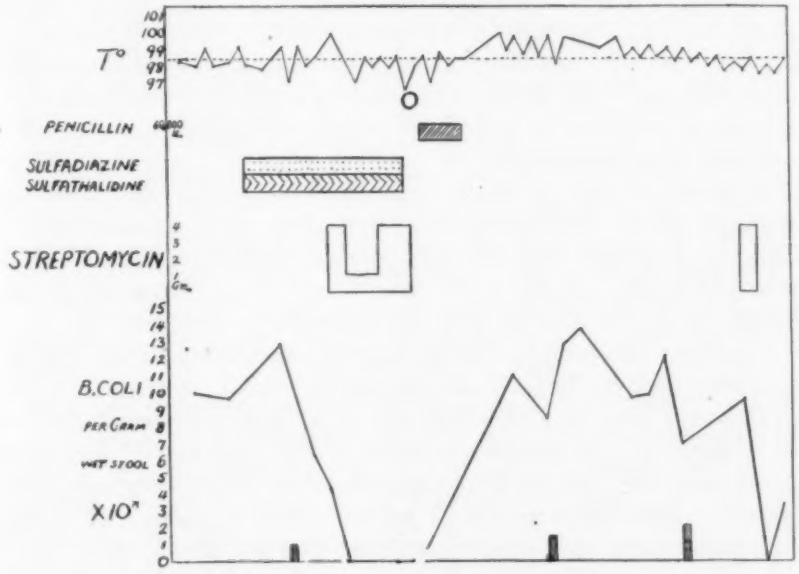

Chart of typical response to oral streptomycin therapy.

was judged by counting shake cultures of serial dilutions of wet feces. It was found that in half the cases there was no growth of Escherichia coli in the cultures. We then increased the dose by mouth to 2 Gm. a day and obtained a slightly higher bacteriostatic effect. Unfortunately, the antibiotic had no pronounced clinical effect on the ulcerative colitis, but the bacteriologic results suggested its possible preoperative value in colon surgical procedures. We have, therefore, been using it since then solely by mouth before operations on the intestines.

2. Morton, H. S., and Smith, F.: Topical Application of Streptomycin in Peritonitis, Arch. Surg. **56**:538 (April) 1948. Morton, H. S.: Streptomycin in Surgery, Canad. M. A. J. **58**:227, 1948.

In the majority of cases, 2 Gm. of streptomycin by mouth per day will give virtually sterile cultures of the feces after forty-eight hours, and the patient is then ready for operation. But in obstruction, and in some cases of diarrhea, this response does not occur. The work of Thatcher and MacLean [3] on the synergistic action between the sulfonamide drugs and streptomycin against gram-negative bacteria suggested that both of these drugs be used. We then tried giving phthalylsulfathiazole or succinylsulfathiazole [4] for a week before operation, with the same dose of streptomycin for forty-eight hours. This improved the response, but there were still some failures. We then gave phthalylsulfathiazole and 4 Gm. of streptomycin, but even this failed in 4 cases of obstruction and in 1 case of diarrhea.

In obstruction the drug has no opportunity to reach the site, being dammed back by the obstinate contents. This is a simple mechanical explanation of our failures, but the only way of testing its validity is to do filtrates of the contents of the bowel and measure the concentration of streptomycin in them. This we are now doing, but it is too recent to give any definite results.

The results of orally administered streptomycin described by Rowe, Spaulding, Bacon and Madajewski [5] were apparently satisfactory with 2 Gm. doses daily combined with phthalylsulfathiazole, while Zintel, Ravdin and Wiley [6] reported a relative effectiveness of streptomycin and stated that 95 per cent of the drug is recoverable in the feces.

In the case of diarrhea, the patient was producing over 4,000 cc. of excreta per day. In spite of this dilution, the concentration of the drug was still in excess of 1,000 units per cubic centimeter and the most resistant organism was sensitive to streptomycin in 320 units per cubic centimeter, but still we were unable to reduce the count below 1,000,000. This was interpreted as too quick a passage to enable the streptomycin to have time for its bacteriostatic effect.

So far, there have been no toxic effects from the oral administration of streptomycin, but the prolonged use is not recommended, for one of our colleagues used the drug for two weeks, 2 Gm. daily, and found that fever, malaise and a smooth glossy tongue developed, frank

3. Thatcher, F. S., and MacLean, J. T.: Synergistic Action Between the Sulfonamides, Certain Dyes, and Streptomycin Against Gram-Negative Bacetria: Preliminary Report, J. Urol. **57**:902, 1947.

4. Hunt, A. H.: Use of Medicaments in Diseases of the Colon, Proc. Roy. Soc. Med. **39**:549, 1946.

5. Rowe, R. J.; Spaulding, E.; Bacon, H. E., and Madajewski, D.: Evaluation of Oral Streptomycin as an Adjunct in Preparing the Large Bowel for Surgery, Bull. Am. Coll. Surgeons **32**:244, 1947.

6. Zintel, H. A.; Ravdin, I. S., and Wiley, M.: The Relative Effectiveness of Oral Streptomycin, Sulfathalidine and Succinylsulfathiazole in Reducing the Bacterial Content of the Feces, Bull. Am. Coll. Surgeons **32**:244, 1947.

evidence of riboflavin deficiency. Treatment with the drug was stopped, and large doses of vitamins and glycerine capsules containing normal feces were given to restore the intestinal flora. This is probably unnecessary, as after stopping treatment with the drug, and with ordinary food, we have observed that the normal flora return both quantitatively and qualitatively within forty-eight hours, although we have as yet no knowledge of whether such restored flora possess the original vitamin synthetic ability.

CONCLUSIONS

The conclusions we have now reached are that with diarrhea, such as in patients with ulcerative colitis, we recommend that the patient be given 4 Gm. of phthalylsulfathiazole daily for one week, as we have observed that sulfonamide drugs given before the antibiotic prevented the development of streptomycin fastness, and 4 Gm. of streptomycin daily for three days, the stool cultures being checked before operation in order to confirm the bacteriostatic effect. In cases without diarrhea the same dose of phthalylsulfathiazole is given, but only 2 Gm. of streptomycin daily, for three days. When there is obstruction, this condition must first be relieved, and then irrigations containing 1 Gm. of streptomycin should be introduced, both from above and below. With these methods we expect to produce a clean field for operation in over 85 per cent of cases.

DISCUSSION

Col. F. E. HAMILTON, Washington, D. C.: I wish to congratulate Dr. Morton on his presentation of streptomycin in gastrointestinal surgery. Streptomycin has been used in similar fashion in army installations.

I wish to discuss 41 consecutive unselected patients operated on for some type of gastrointestinal disorder during the year 1947 at Walter Reed Hospital, Washington, D. C. Included are thirty operations on the colon and eleven operations on the stomach and small intestine. Preoperative preparation consisted of 0.5 Gm. of streptomycin by mouth every six hours for four days. In this series, streptomycin alone was used. In addition, preoperative measures were taken to maintain a proper fluid and electrolyte balance along with a mechanically clean gastrointestinal tract. At operation, all of the intestinal resections were of the open type. Gross contamination was avoided, but no attempt was made to avoid bacteriologic contamination. Smears taken from the open intestinal lumen at operation showed a small number of colonies of apparently attenuated bacteria.

There were no serious postoperative complications in this series. There were no deaths and no instances of peritonitis. The only complications consisted of two minor wound infections. In addition, there was no evidence of mucosal irritation or intestinal hemorrhage, no alteration of prothrombin levels and no vestibular disturbance when the streptomycin was administered for a relatively short period of time, as in these cases.

Management of these patients prepared with streptomycin requires the usual adherence to the principles of atraumatic surgery. The advantage to be gained is an increased safety factor. By reduction of the operative hazard, certain major surgical procedures are greatly simplified.

INGUINAL AND FEMORAL HERNIOPLASTY: ANATOMIC REPAIR

CHESTER B. McVAY, M.D., Ph.D.
YANKTON, S. D.

IT IS the purpose of this brief communication to present a concept of the anatomic defect that exists in the various types of groin hernias and to outline a method for their correction which is in essence a restoration of the inguinofemoral structure to normal. In as much as Dr. Barry J. Anson and I, in collaboration, have repeatedly presented the detailed anatomy of the region,[1] no attempt will be made here to review these papers. As a matter of fact, I presented a paper dealing with the anatomy of inguinal and femoral hernias before the first meeting of the Central Surgical Association in 1941 [1b] and pointed out the error in using the inguinal ligament in any part of the repair of an inguinal or femoral hernia. In the ten years since Dr. Anson and I first published our anatomic observations and suggested a change in the conventional methods of dealing with inguinal hernias, several surgeons have published papers dealing with the advisability of using Cooper's ligament in place of the inguinal ligament in the repair of inguinal hernias.[2] Using the method to be described, I now have a series of

---

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

From the Department of Anatomy, Northwestern University Medical School, Chicago, and the Department of Surgery, University of South Dakota Medical School and the Yankton Clinic, Yankton, S. D.

1. Anson, B. J., and McVay, C. B.: (*a*) The Anatomy of the Inguinal and Hypogastric Regions of the Abdominal Wall, Anat. Rec. **70**:211-225, 1938; (*b*) Inguinal Hernia: I. The Anatomy of the Region, Surg., Gynec. & Obst. **66**:186-191, 1938; (*c*) Fascial Continuities in the Abdominal Perineal and Femoral Regions, Anat. Rec. **71**:401-407, 1938; (*d*) Aponeurotic and Fascial Continuities in the Abdomen, Pelvis and Thigh, ibid. **76**:213-231, 1940; (*e*) Composition of the Rectus Sheath, ibid. **77**:213-225, 1940; (*f*) A Fundamental Error in Current Methods of Inguinal Herniorrhaphy, Surg., Gynec. & Obst. **74**:746-750, 1942; (*g*) McVay, C. B.: A Fundamental Error in the Bassini Operation for Direct Inguinal Hernia, Univ. Hosp. Bull. Ann Arbor **5**:14, 1939; (*h*) An Anatomic Error in Current Methods of Inguinal Herniorrhaphy, Ann. Surg. **113**:1111-1112, 1941; (*i*) Inguinal and Femoral Hernioplasty, read before the Minneapolis Surgical Society, Nov. 6, 1947.

2. (*a*) Farris, J. M.: Some Technical Aspects of the McVay Herniorrhaphy, read before the Society of University Surgeons, New Orleans, Jan. 31, 1948. (*b*) Harkins, H. N.; Szilagyi, D. E.; Brush, B. E., and Williams, R.: Clinical

one hundred inguinal and femoral hernioplasties (from one to nine years since operation) with no recurrence, although it must be acknowledged that it has been impossible to trace 4 of the cases.

ABDOMINAL RING REPAIR

It is generally recognized that the defect in a small indirect inguinal hernia is the presence of a congenital peritoneal diverticulum through the abdominal inguinal ring that extends for a variable distance intimately associated with the cord structures and within the internal spermatic fascia. The size of the abdominal inguinal ring and the strength and consistency of the posterior inguinal wall and the status of the femoral ring and canal can be accurately determined by a combination of intraperitoneal palpation through the opened hernial sac and inspection anteriorly (the spermatic cord is always elevated from its bed by incising the delicate cremaster fascia, so that the posterior wall can be properly appraised). If the abdominal inguinal ring is not significantly dilated, if a femoral hernia does not exist and if there is no weakness or herniation of the posterior inguinal wall, then the only problem is the extirpation of the hernial sac and closure of the abdominal inguinal ring. The indirect hernial sac is dissected free from the cord structures well above the level of the fascial abdominal inguinal ring, ligated high and excised in the conventional manner. The slightly dilated abdominal inguinal ring is then closed snugly about the spermatic cord by one or two interrupted medium silk mattress sutures which approximate the transversalis fascia to the anterior layer of the femoral sheath, medial to the cord (fig. 1). This completes the repair of this simple congenital type of inguinal hernia. The spermatic cord is dropped back into normal position against the posterior inguinal wall and the external oblique aponeurosis is closed over it with interrupted fine silk sutures, making a snug subcutaneous inguinal ring in the natural position.

RECONSTRUCTION OF THE POSTERIOR INGUINAL WALL

The posterior inguinal wall is the lowermost portion of the transversus abdominis aponeurosis; its insertion is into Cooper's ligament, and it is roughly outlined by the classic delineation known as Hessel-

---

Experiences with the McVay Herniotomy, Surgery **12**:364-377, 1942. (*c*) Harkins, H. N., and Swenson, S. A., Jr.: Cooper's Ligament Herniotomy: Clinical Experience in Three Hundred and Twenty-Two Consecutive Cases, S. Clin. North America **23**:1279-1297, 1943. (*d*) Lich, R., and Samson, R. B.: Method of Inguinal Herniorrhaphy, J. Internat. Coll. Surgeons **8**:454-458, 1945. (*e*) Mattson, H.: Use of Rectus Sheath and Superior Pubic Ligament in Direct and Recurrent Inguinal Hernia, Surgery **19**:498-503, 1946. (*f*) Rice, C. O., and Strickler, J. H.: The Repair of Hernia with Special Application of the Principles Evolved by Bassini, McArthur and McVay, Surg., Gynec. & Obst. **86**:169-176, 1948.

bach's triangle. The transversalis fascia is closely adherent to its posterior surface, and a thinner fascial layer is equally adherent to the anterior surface of this aponeurosis. The strength of the posterior wall is directly proportional to the number of transversus abdominis aponeurotic fibers that it contains. It is the primary purpose of this paper to demonstrate that the large indirect inguinal, the direct inguinal and the femoral hernia are all either encroachments on or defects in this all-important posterior inguinal wall.

The large indirect inguinal hernia encroaches on the posterior inguinal wall from the lateral side, pushing the inferior epigastric vessels medially, and the destruction of the posterior inguinal wall varies dirctly as the diameter of the abdominal inguinal ring. In the very large variety of indirect inguinal hernia where the medial wall of the neck of the hernial sac lies against the tendon of the rectus abdominis

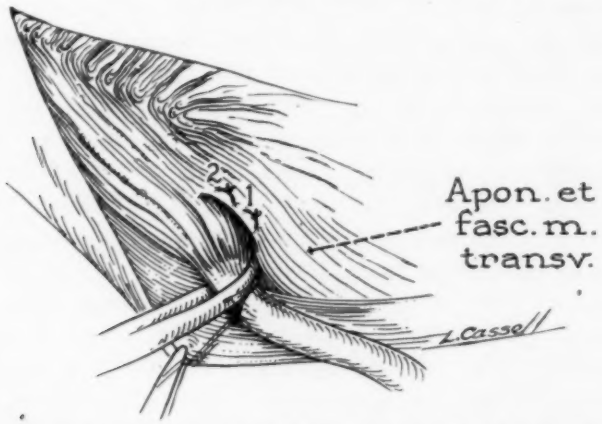

Fig. 1.—The indirect inguinal hernial sac has been excised and the slightly dilated abdominal inguinal ring closed snugly, medial to the cord by a single mattress silk suture (1) which approximates the transversalis fascia to the anterior layer of the femoral sheath. Suture 2 approximates the fascia of the cord to the transversalis fascia. The inguinal ligament has been pulled inferiorly to demonstrate the continuity of the transversalis fascia and the anterior layer of the femoral sheath.

muscle, the only portion of the posterior wall that remains is a small triangular piece in the angle between Cooper's ligament and the rectus abdominis tendon of origin. Thus, depending on the size of the dilated abdominal inguinal ring, either all or part of the posterior inguinal wall must be reconstructed. After the hernial sac is dealt with in the conventional manner, all of the attenuated aponeuroticofascial posterior inguinal wall is excised, and after making the relaxing incision (Rienhoff [3]) the reconstruction of the posterior inguinal wall is started.

3. Rienhoff, W. F.: The Use of the Rectus Fascia for Closure of the Lower or Critical Angle of the Wound in the Repair of Inguinal Hernia, Surgery **8**:326-339, 1940.

The direct inguinal hernia is a defect directly through the posterior inguinal wall, and the defect varies in size from the small pedunculated type to the large bulging type which destroys the entire posterior inguinal wall. In the bulging type, all weakened aponeuroticofascial components of the posterior inguinal wall are excised from the abdominal inguinal ring to the pubic tubercle. In the pedunculated type, no attempt is made to simply close the small opening, but all of the posterior wall inferior to the opening and down to Cooper's ligament is excised. Thus, at this point the problem of repair is the same for the two varieties of direct inguinal hernia, but there is usually more of the posterior inguinal wall remaining in the pedunculated type. The relaxing incision

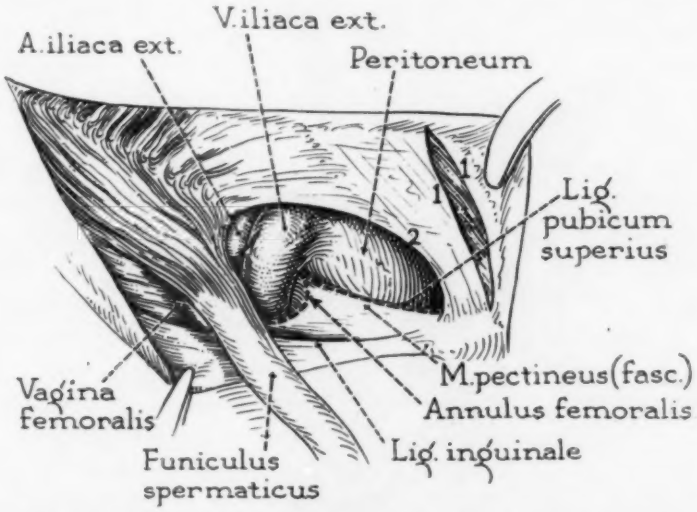

Fig. 2.—The weakened portion of the posterior inguinal wall has been excised, leaving a strong superior margin to be used in the repair (2). In a femoral hernia the defect is much smaller. The relaxing incision has been made (1). Cooper's ligament (ligamentum pubicum superius) is clearly defined and the inguinal ligament retracted inferiorly to demonstrate the pectineus fascia and the femoral sheath. The dotted line along Cooper's ligament and around the femoral vein up onto the anterior femoral sheath represents the line along which the new posterior wall will be sutured.

(Rienhoff [3]) is made, and the reconstruction of the posterior inguinal wall is started.

The femoral hernia is a defect in the posterior inguinal wall by virtue of the narrowing of the insertion of the posterior inguinal wall into Cooper's ligament. Whether the presence of a femoral hernia is due to a congenitally narrow insertion or whether the femoral hernia gradually attenuates the lateralmost edge of the insertion of the posterior inguinal wall into Cooper's ligament, the fact remains that the object

of the repair must be to broaden the attachment of the posterior inguinal wall into Cooper's ligament. After elevation of the spermatic cord from its bed by incision of the cremaster fascia, the posterior inguinal wall is incised close to Cooper's ligament, if there is no concomitant weakness of the wall as in a direct inguinal hernia. This incision extends from near the pubic tubercle to the femoral vein and up over the vein to the abdominal inguinal ring by incision of the transversalis fascia at the line along which it becomes the anterior femoral sheath. The neck of the femoral hernia is identified and the hernia pulled through the femoral ring, and the inguinal ligament cut if necessary. The femoral hernial sac which is now in the position of a direct inguinal hernia, is opened, inspected, ligated and excised. The relaxing incision is made, and the reconstruction of the posterior inguinal wall is started.

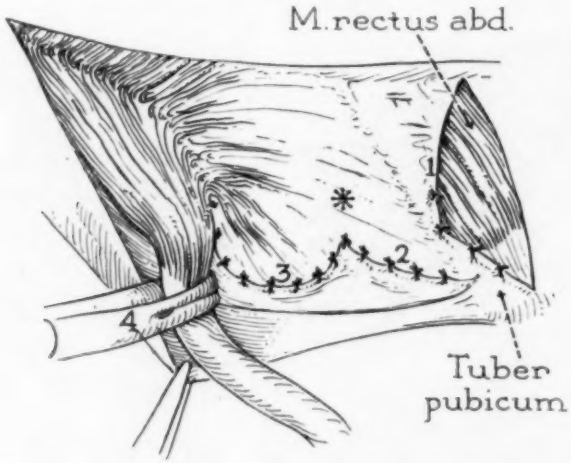

Fig. 3.—The reconstruction is complete. The previously incised transversus stratum has been sutured to Cooper's ligament from pubic tubercle to femoral vein (2) and then to the anterior femoral sheath (3). The cord has been pulled laterally (4) to demonstrate the closure of the abdominal inguinal ring. The relaxing incision is now a triangular defect, and the lateral margin has been sutured to the underlying tendon of the rectus abdominis muscle. The spermatic cord is dropped in against the new posterior inguinal wall and the external oblique aponeurosis closed over it with the subcutaneous inguinal ring in the normal position.

It will be readily noted that the end result of the preparation for the reconstruction of the posterior inguinal wall is the same (fig. 2) in the large indirect inguinal, the direct inguinal and the femoral hernia. In all three types there is a strong free margin of posterior inguinal wall above by virtue of the removal of all thinned out fascial and aponeurotic components. Cooper's ligament is cleaned of fascial and aponeurotic tags, and the preperitoneal connective tissue is held

out of the operative field with a small ribbon retractor. The femoral vein is exposed to avoid inadvertent injury, and the margin of the femoral sheath is freed up into the abdominal inguinal ring. The previously incised margin of the transversus layer (the posterior inguinal wall) is then sutured to Cooper's ligament from pubic tubercle to the femoral vein by a series of interrupted medium silk sutures (fig. 3 [2]). This reattaches the posterior inguinal wall to its normal insertion and even broadens this insertion, because the femoral ring is obliterated. The suture line then comes up over the femoral vein, approximating the transversus layer to the anterior layer of the femoral sheath (fig. 3 [3]); the first of these sutures includes the pectineus fascia. At about this point the posterior wall becomes muscular, and the sutures therefore incorporate only the transversalis fascia. This suture line is continued, with interrupted medium silk sutures, to the abdominal inguinal ring which is snugly closed about the spermatic cord (fig. 3). This completes the repair. It will now be noted that the relaxing incision in the rectus sheath is a triangular defect through which the lower portion of the rectus abdominis muscle and its tendon of origin are seen (fig. 3). The lateral retracted edge of this relaxing incision is usually sutured to the underlying rectus tendon (fig. 3 [1]). It is now apparent that the new posterior inguinal wall is a combination of the remaining portion of the old posterior inguinal wall and the rectus sheath. The transfer has been accomplished by a process of sliding, and the direction of the aponeurotic fibers in the new posterior wall is the same as in the normal undisturbed inguinal region. This is certainly more anatomic and, it seems to me, more physiologic than the turning of a flap of the rectus sheath after the method of the original Bloodgood operation or as more recently advocated by Farris,[2a] Mattson [2e] and others. After observing this operative procedure, surgeons have often questioned me about a weak area along the lateral retracted edge of the rectus sheath incision. If one raises the lateral margin of the relaxing incision, he will note that the bulk of the transversalis fascia leaves the transversus aponeurosis at the lateral border of the rectus muscle and invests that muscle (the rectus fascia). Thus the protrusion of preperitoneal fat is prevented by this fascial layer and the suturing of the sheath to the rectus abdominis tendon of origin.

After the reconstruction of the inguinal wall, as previously outlined, is completed, the spermatic cord is dropped in against the new posterior inguinal wall and the external oblique aponeurosis is closed over it with interrupted fine silk sutures, making a snug subcutaneous inguinal ring in the normal position. Subcutaneous fascia and skin are closed with interrupted fine silk sutures.

SUMMARY

Abdominal ring repair is a simple procedure used for all small uncomplicated indirect inguinal hernias after excision of the hernial sac. There is no anatomic defect in the posterior inguinal wall, and no additional repair is necessary or advisable.

Reconstruction of the posterior inguinal wall is a procedure used for large indirect inguinal, direct inguinal and femoral hernias or any combination of groin hernias. It is demonstrated that these three types of hernia are all defects in the posterior inguinal wall (the transversus abdominis aponeurosis and fascia) and therefore logically require the same reconstructive procedure. This repair is a restoration of the inguinal anatomy to normal.

GLOMUS TUMORS

A Summary of Fifteen Cases

KENNETH E. LEMMER, M.D.
MADISON, WIS.

THE EXISTENCE of neoplasms of the neuromyoarterial glomus has been recognized for many years, and they have been variously called angiosarcomas, glomangiomas or hemangiomatous tumors. Little has been written in the ordinary textbooks about these lesions, and it is only by a careful review of the current literature that a true picture of these interesting tumors can be obtained. They are relatively rare lesions which contain vascular, epithelial and nervous elements and for the most part are located in the cutaneous or subcutaneous tissues. Glomus tumors have also been described as occurring in the knee joint and in the muscles and tendons where normal glomuses have not been identified. They are benign in their behavior, with little or no tendency to recur if they are completely removed, although Murray and Stout [1] have reported a glomus tumor which displayed progressive infiltrative growth. Wood,[2] in 1812, first gave a clinical description of these lesions, calling them painful subcutaneous tubercles, but probably included many cases which were not glomus tumors. Later, in 1924, Masson [3] first pointed out that these tumors originated in the cutaneous glomus and correlated the clinical and microscopic observations in these cases. The normal architecture of the neuromyoarterial glomus was carefully investigated by Popoff,[4] who, using serial sections, described the structure as consisting of an efferent

---

From the Department of Surgery, University of Wisconsin School of Medicine, Madison, Wis.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

Mr. John Mullins, Department of Pathology, and Mr. Homer Montague, Department of Photography, aided in the preparation of sections, staining and photomicrography.

1. Murray, M. R., and Stout, A. P.: The Glomus Tumor, Am. J. Path. **28**:183-203 (March) 1942.

2. Wood, W., cited by Theis, F. V.: Subungual Neuromyoarterial Tumor of the Toe, Arch. Surg. **34**:1-11 (Jan.) 1937.

3. Masson, P.: Le glomus neuromyoarterial des regions tactiles et ses tumeurs, Lyon chir. **21**:257-280 (May-June) 1924.

4. Popoff, N. W.: The Digital Vascular System, Arch. Path. **18**:295-330 (Sept.) 1934.

arteriole from which branches a tortuous anastomotic vessel (the Sucquet-Hoyer canal), which in turn opens directly into a primary collecting vein. As he pointed out, the anastomotic vessel is endothelial lined and surrounded by longitudinal and circular smooth muscle fibers, among which appear the glomal cells, which are epithelioid in appearance with oval or globular nuclei. The exact origin of these glomal cells has not been determined, and they have been described variously as angioblasts, embryonic muscle cells or elastoblasts. In

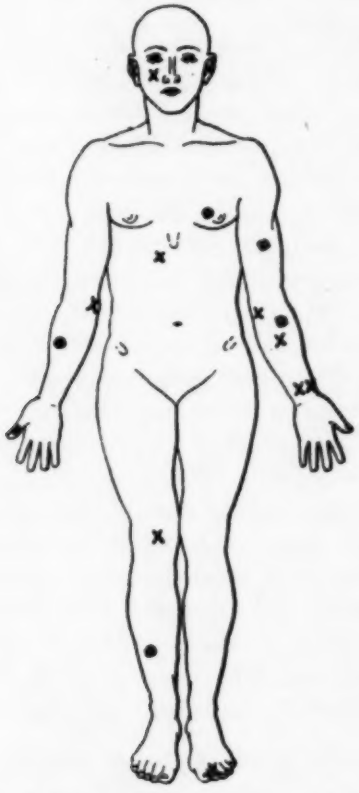

Fig. 1.—Distribution of glomus tumors (the crosses indicate point of occurrence in male subjects and the dots in female subjects).

addition, there is a delicate collagenous reticulum which binds the glomus together and in which run the fine nonmyelinated nerve fibers. The normal glomus does not exceed 1 mm. in diameter and is thought to be a shunt between the arterial and venous system without the interposition of capillaries. This is also connected with the autonomic nervous system, which may explain the extreme pain, temperature changes and sweating that often occur as a result of the tumor. The normal glomus probably has a large role in the control of body tempera-

ture, and this arteriovenous shunt may act to send more blood to the surface to aid in heat dispersion in high temperatures and to divert the blood from the surface to prevent heat loss in low temperatures. It has been pointed out by Beaton and Davis [5] that the glomus is absent in newborn infants, which may explain their lack of ability to regulate the body temperature.

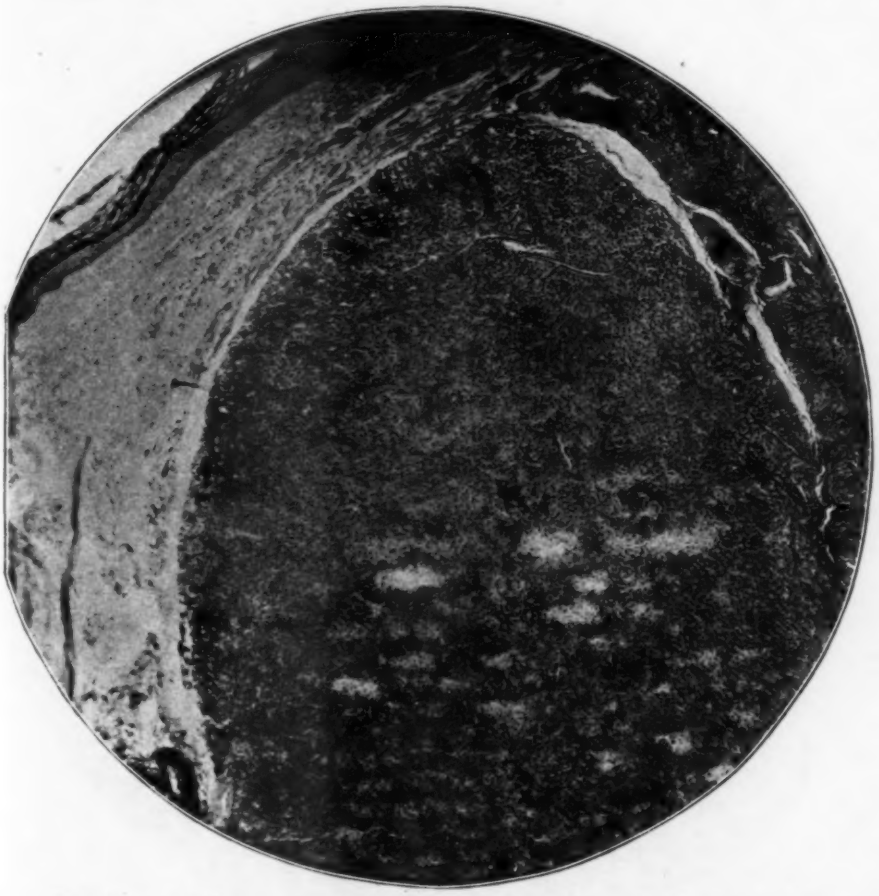

Fig. 2.—A solid tumor containing large numbers of glomal cells with few vascular channels and few nerve fibers.

The clinical picture of a glomus tumor is as follows: The patient usually complains of a small subcutaneous nodule which is bluish or purple in color, which may be present under the nail but also may be found anywhere on the body surface. At first the tumor may not

5. Beaton, L. E., and Davis, L.: Glomus Tumor: Report of Three Cases; Analysis of Two Hundred and Seventy-One Recorded Cases, Quart. Bull., Northwestern Univ. M. School **15**:245-254, 1941.

be painful. If pain is present, it usually radiates in a peripheral direction from the tumor, and an increase in the pain may be noted on exposure to heat or cold or by changes in position or pressure. Theis [6] expressed the belief that the pain is due to pressure on the component nerves, as it has been observed that these tumors often enlarge in size during attacks of pain. The appearance of increased

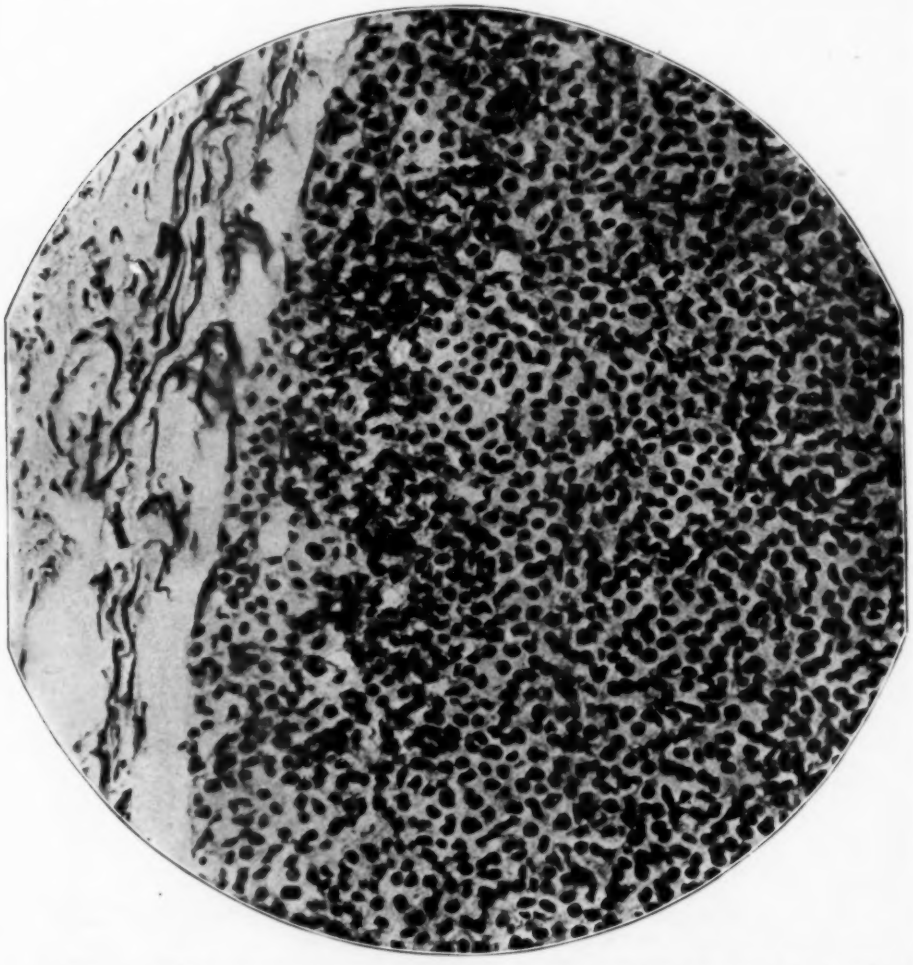

Fig. 3.—High power photomicrograph shows the decided increase of glomal cells.

sweating, either localized over the tumor or in the area of distribution of pain, has also been noted. This may be associated with an increased temperature of the same area.

6. Theis, F. V.: Subungual Neuromyoarterial Tumor of Toe: Effect of Increased Peripheral Temperature, Arch. Surg. **34**:1-11 (Jan.) 1937.

In the early reports most of the tumors were located in the extremities and particularly under the nails. However, Stout [1] in 1935 noted that the subungual and finger tip locations were commoner in women and the other situations on the body in men. This was well illustrated in an excellent review article by Beaton and Davis,[5] in which the distribution of single glomus tumors was charted. Loeb,[7] in reviewing the literature

Fig. 4.—A vascular type of tumor containing glomus cells surrounded by vascular spaces.

in 1939, was able to collect 144 cases of glomus tumor in the course of his studies to determine the relationship between trauma and the formation of these lesions. He stated the belief that only about 20

7. Loeb, M. J.: Trauma and Glomus Tumors, Indust. Med. **10**:208-213 (May) 1941.

per cent were due to injuries, whereas other authors estimated that up to 50 per cent had been preceded by definite trauma.

From the time of Loeb's [7] report in 1939, at which time he recorded 144 cases, until the report by Beaton and Davis [6] in 1941, an additional 127 cases had been described in the literature. This relative increase was probably due to a greater interest in the subject rather than to

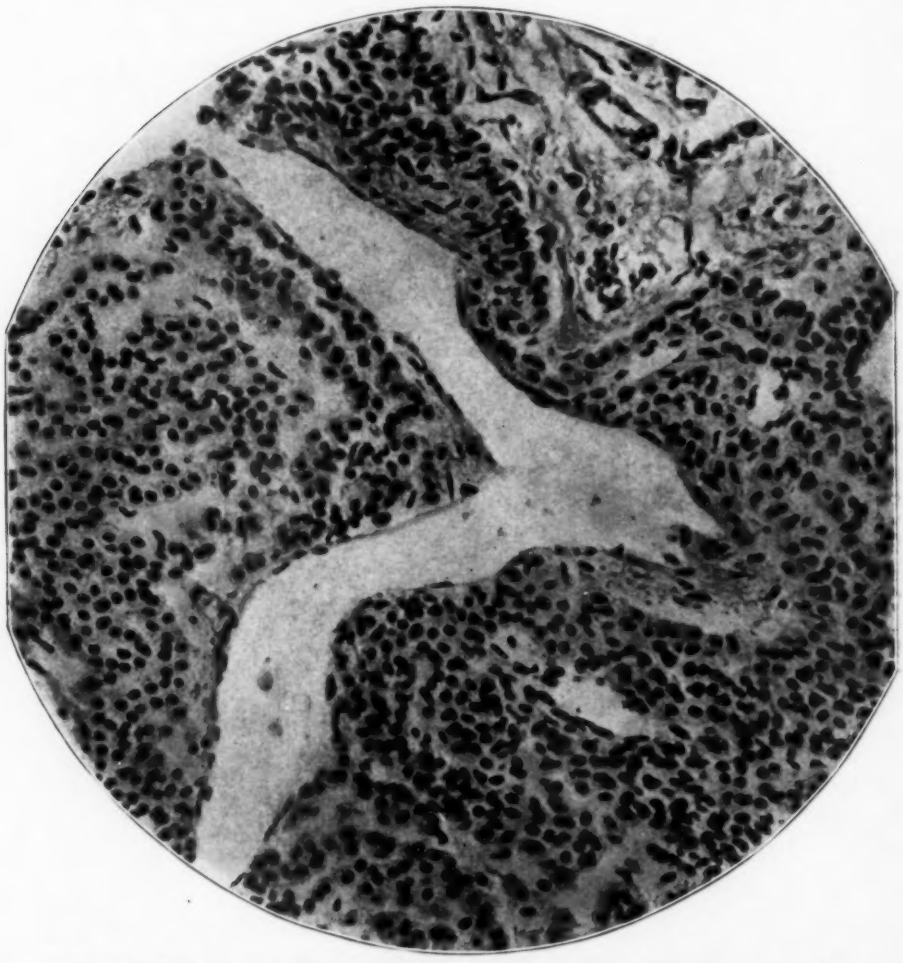

Fig. 5.—High power photomicrograph of a section of the tumor a section of which is shown in figure 4.

an actual increase in the number of tumors. Recent reports indicate that multiple painful glomus tumors occasionally exist in the same person, and Weidman and Wise [8] in 1937 described a woman having

8. Weidman, F. D., and Wise, F.: Multiple Glomus Tumors of Order of Telangiectasis, Arch. Dermat. & Syph. **35**:414-426 (March) 1937.

forty-eight painless glomus tumors. Later, Slepyan [9] reported a case with twelve such painless tumors associated with two typical painful lesions, so that the typical clinical syndrome of a glomus tumor may have to be revised. Kaufman and Clark [10] reported 4 cases of glomus tumor in the same family; one member had multiple lesions. They, too, feel that the lesion may not be as uncommon as supposed but that unfamiliarity

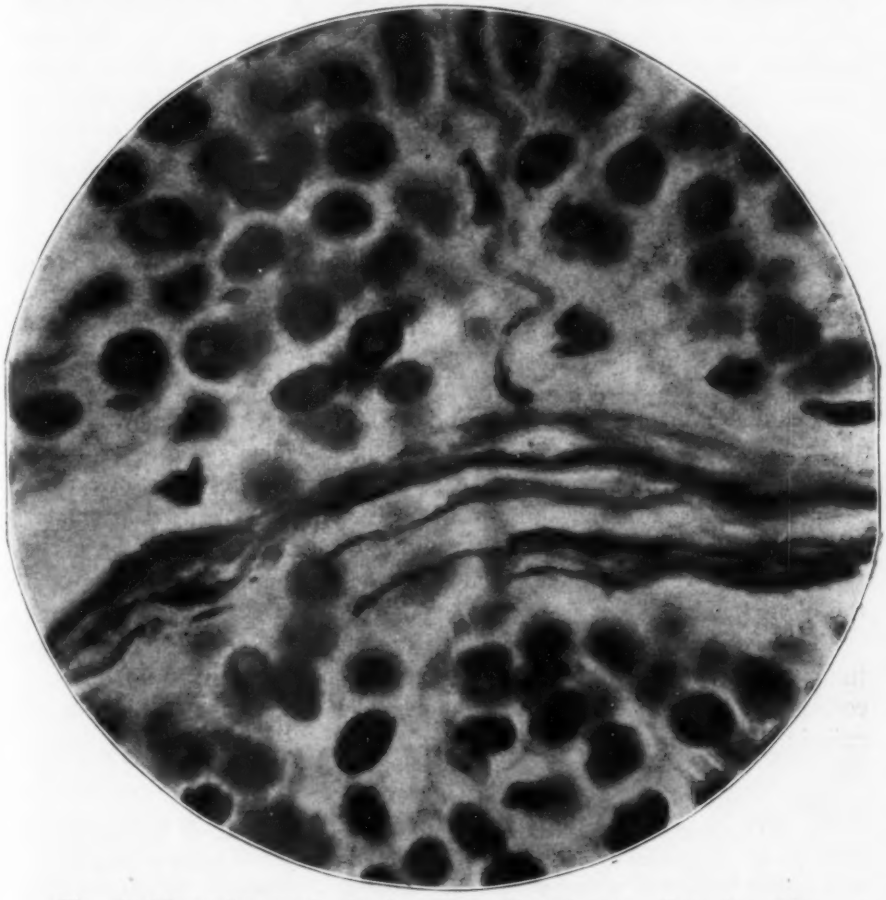

Fig. 6.—Photomicrograph showing nonmyelinated nerve fibers branching out between the glomal cells.

with the conditions by clinicians may have caused many tumors to be overlooked or incorrectly diagnosed.

The appearance of 2 cases of glomus tumor within a relatively short time led me to review the cases at the Wisconsin General Hospital and

9. Slepyan, A. H.: Multiple Painful and Painless Glomus Tumors, Arch. Dermat. & Syph. **50**:179-182 (Sept.) 1944.

10. Kaufman, L. R., and Clark, W. T.: Glomus Tumors: Four Cases in Same Family, Ann. Surg. **114**:1102-1105 (Dec.) 1941.

the State Laboratory of Hygiene. I was interested in learning what percentage of the tumors were painful, whether there were multiple tumors in any of our cases and whether the clinical observations could be associated with a definite type of pathologic picture. In the 15 cases here summarized, seven of the tumors were definitely painful and three nonpainful, and 5 patients complained of slight tenderness with pressure over the tumor. No multiple tumors were seen. Nine of the tumors were found in male subjects and 6 in female. The age incidence varied from 22 to 85 years of age, with an average of 56 years of age. The distribution of the lesions is shown in figure 1. The duration of tumors was from six months to twenty-fours years, an average of fifteen years. The lesions were rarely diagnosed by the clinician, and the commonest erroneous diagnoses were those of neuroma, fibroma and neurofibroma.

The microscopic picture closely resembles the normal glomus except for a decided increase in the glomal cells and nonmyelinated nerve fibers. Two main types of lesions may be found: One (fig. 2.), a solid tumor, contains large numbers of glomal cells with few vascular channels and few nerve fibers; figure 3 (in high power) shows the decided increase of glomal cells and two (fig. 4), a vascular type of tumor, contains glomus cells surrounded by vascular spaces. Figure 5 (high power) is a section of the same tumor. These sections were stained with hematoxylin and eosin. Silver stains were used in similar sections, including Bodian and phosphotungstic acid, to show the nerve fibers. Figure 6 shows nonmyelinated nerve fibers branching out between the glomal cells.

There appear to be more nonmyelinated fibers present in the vascular tumors than in the highly cellular tumors, but there was no obvious correlation between the type of tumor and the presence or absence of pain in the patient.

DISCUSSION

DR. J. GRAFTON LOVE, Rochester, Minn.: As neurosurgeons we are sometimes called on to see glomus tumors. Although this is one of the smallest tumors we have to deal with, it is a very painful and disabling condition. It is too bad that occasionally someone loses an arm or leg because of this tumor. Concerning the microscopic picture, the pathologist is apt to consider this a malignant tumor. However, a diagnosis usually can be made clinically by the pin test. These patients are much like patients with trigeminal neuralgia. If a pin is pressed near the lesion the pain will not occur; yet if the lesion is touched the patient will have excruciating pain. This is a typical observation and aids in a correct diagnosis. Treatment should be by excision with local anesthesia, and the local anesthetic should be given by the operating surgeon because the lesion is so small it may be otherwise overlooked.

KELOIDS AND HYPERTROPHIC SCARS

HAROLD M. TRUSLER, M.D.
AND
THOMAS B. BAUER, M.D.
INDIANAPOLIS

AN UNSIGHTLY scar is often a matter of concern to a patient who acquires such a disfigurement. For this reason, if for no other, it should be a matter of concern to the surgeon. Perhaps the commonest causes of scar disfigurement are hypertrophy of scar tissue and keloid formation. The two conditions are frequently confused, and it is often difficult to differentiate them. The chief differences are as follows: (1) The hypertrophic scar remains confined to the area of skin incision or injury; the keloid tends to invade the surrounding skin. (2) The hypertrophic scar continues to grow for several weeks or months, then becomes inactive and tends to regress; the keloid, however, continues its growth into bizarre conglomerate forms which do not regress. In fact, that is how it derives its name, keloid being a derivation of the Greek word meaning claw. Though these clinical differences are obviously important, it is difficult in the early stages of scar hypertrophy to determine whether the proliferation of scar tissue will be confined to the incision or area of trauma or whether it will continue its growth to become a progressive keloid.

PATHOLOGIC ASPECTS

Microscopically it is difficult to distinguish the simple hypertrophic scar from the true keloid. In each there is an overgrowth of scar tissue composed of normal-appearing fibroblastic cells. The stimulus producing the overgrowth seems to be some form of trauma to the dermis. Trauma to the epidermis alone will not produce this response. This is graphically illustrated by the fact that keloids and scar hypertrophy often occur after deep second and third degree burns, almost never after first degree burns.

Keloidal growths represent the twilight zone between normal cell proliferation and neoplastic growth. The direction of the keloid invasion is into the surrounding skin and to only a limited extent into the

From the Division of Plastic and Reconstructive Surgery, Indiana University Medical Center.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

subcutaneous tissue. These growths, in the early stages, are hyperemic and cause the patient considerable discomfort because of their tendency to produce sharp pains, itching and burning. In the case of simple scar hypertrophy, after a variable period, usually six months or longer, the hyperemia tends to fade, the discomfort to subside and the scar to flatten out. If there is a progressive keloid tendency, however, the scar does not flatten but remains as a hard tumor mass which may increase to larger proportions as a result of subsequent irritation.

FACTORS IN CAUSATION

Some form of trauma, though it may not always be apparent, is considered to be the exciting factor in the production of all keloids. The fact that some persons respond to trauma with an exaggerated production of scar tissue, while others react with a normal response, has not been fully explained. Numerous theories have been advanced for the development of keloids. Garb and Stone,[1] in 1942, reviewed the literature on this subject and presented a summary of 80 cases of their own. They concluded that hormone stimulation is the "main or important contributing cause of keloid formation." This statement is based on the fact that in their series 34 per cent occurred in the second decade, when hormonal stimulation is high. Spontaneous resolution or absence of keloid was noted after the menopause. Nason[2] postulated "some sort of fibroblastic diathesis varying in intensity among races and individuals." Marshall and Rosenthal[3] stated the opinion that a keloid results as a fibroblastic response to extravasated serum occurring at the site of trauma. They stated the belief that keloids are composed of a considerable amount of edema fluid in addition to fibroblastic tissue. Marshall[4] produced a keloid-like growth on his own thigh by the repeated injections of his own serum into the skin.

Other interesting observations have been made about factors which may have some bearing on the development of keloids and hypertrophic scars. A regional susceptibility to keloid formation has been noted in some persons. In a susceptible person, a scar in one area of the body will become a keloid while a scar in another location may be normal. Keloids and hypertrophic scars may occur any place on the body, but are most commonly found on the sides, back of the neck,

1. Garb, J., and Stone, M. J.: Keloids: Review of Literature and Report of Eighty Cases, Am. J. Surg. **58**:315-335 (Dec.) 1942.

2. Nason, L. H.: Keloids and Their Treatment, New England J. Med. **226**: 883-886 (May 28) 1942.

3. Marshall, W., and Rosenthal, S.: Pathogenesis of Keloids and Similar Neoplasms in Relation to Tissue Fluid Disturbances, Am. J. Surg. **62**:348-357 (Dec.) 1943.

4. Marshall, W.: Fibromatous Skin Lesions Produced by Repeated Blood Serum Injections in the Human, Am. J. Surg. **69**:338-343 (Sept.) 1945.

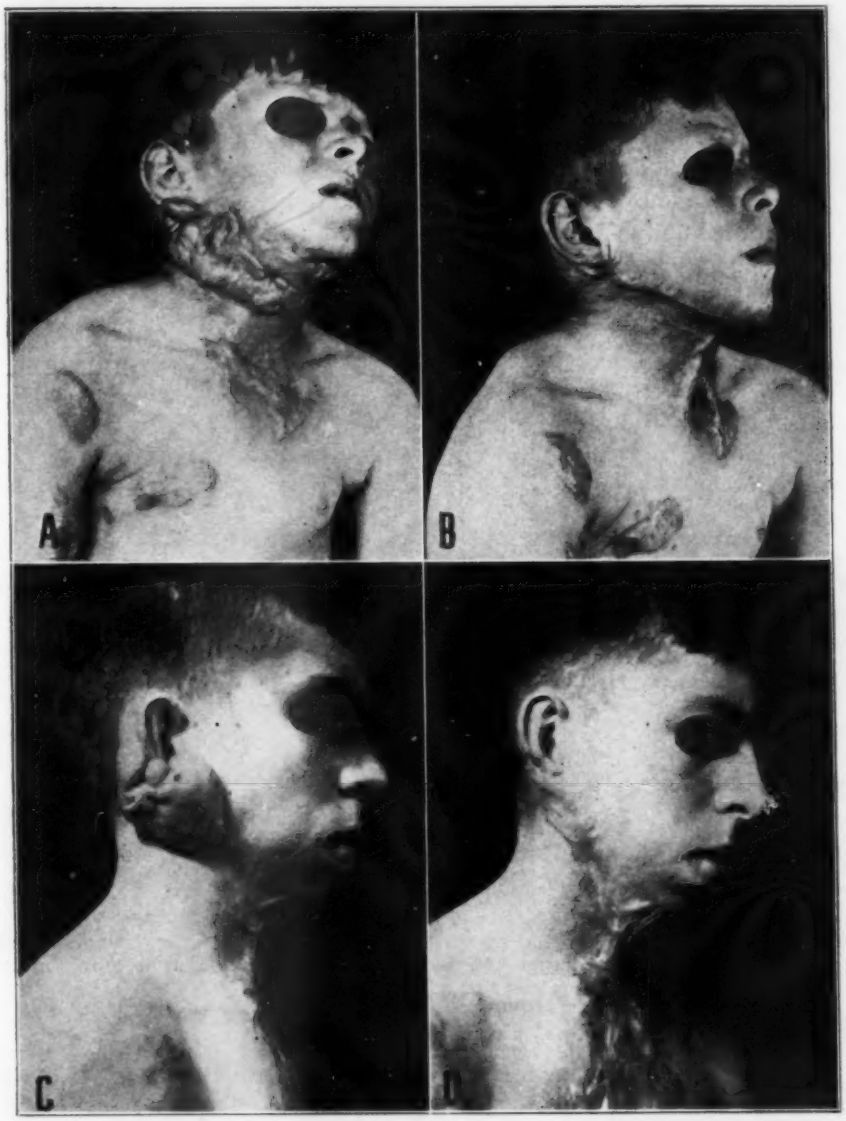

Fig. 1.—Demonstration of a progressive keloid which appeared after a third degree burn of the neck. *A*, the extensive keloid prior to treatment. *B*, appearance after excision of the keloid and closure under some tension. The patient did not return for roentgen therapy or observation. *C*, the same patient five years later, showing massive keloid formation involving the right ear. *D*, view after the keloid had been excised and replaced with a skin graft and roentgen therapy had been given. The pronounced deformity of the chin, which could have been prevented had the contraction been relieved five years earlier, may be noted.

ears, sternal area of the chest, back and abdomen. They are uncommon in areas where the skin tension is constantly changing. It has also been noted that in incisions made in, or parallel to, normal flexion creases keloids do not tend to form while in those which cross the flexion creases these growths are prone to develop. In incisions which are closed under tension, hypertrophic scars are more apt to develop. Third degree burns are frequently followed by the development of keloids. This fact is especially true of those burns that are not skin grafted and in which healing is delayed and occurs by cicatrization. Keloidal growths tend to be hereditary and to appear with unusual frequency in some families. It is a well known fact that keloid formation is much commoner in the Negro race than in the Caucasian races.

Fig. 2.—Small hypertrophic scar of nose which appeared after laceration and which was treated with excision and roentgen therapy. *A*, scar prior to treatment. *B*, appearance after excision and roentgen therapy.

We have been impressed with the observation that hypertrophic scars are more apt to develop in plethoric persons than in patients with a tendency to anemia. In a burned extremity, scars will remain flat until the patient begins to walk or put the foot in the dependent position. Then if there is congestion and edema in the extremity, scar hypertrophy is prone to occur.

TREATMENT

Through the years many ingenious measures and bizarre methods have been advanced for the treatment of keloids, but most of these have been discarded as unsatisfactory. Actually we have no new or

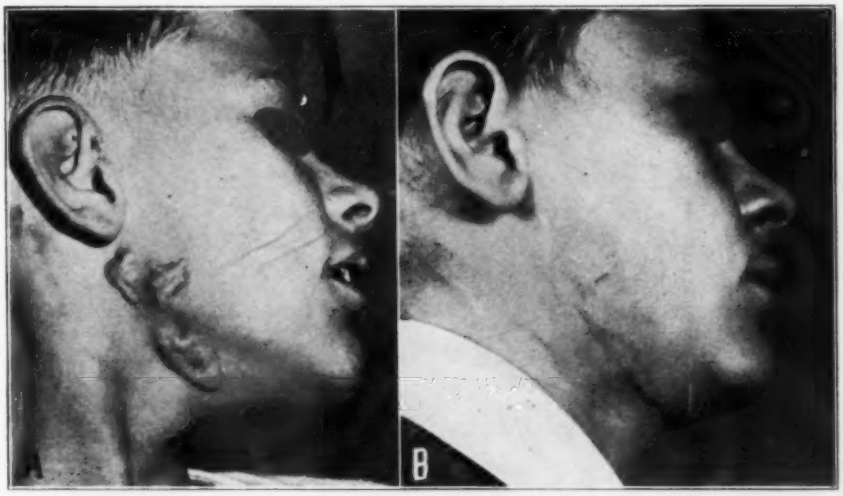

Fig. 3.—Keloids of the neck which resulted from third degree burns occurring more than a year previously. Fractional excision and closure followed by roentgen therapy and pressure were used. *A*, keloid of the neck prior to treatment. *B*, result after initial excision and a course of roentgen therapy. The result can be further improved by an additional excision.

Fig. 4.—Hypertrophic scar of the neck, deformity of the ear and axillary web resulting from an ungrafted third degree burn which had occurred five years earlier. The hypertrophic scar of the neck was treated with excision and multiple Z-plasty. *A*, view of the patient prior to any reconstructive operation, showing heavy "ropelike" scar extending from ear to shoulder. *B*, result six months after correction with one multiple Z-plasty procedure.

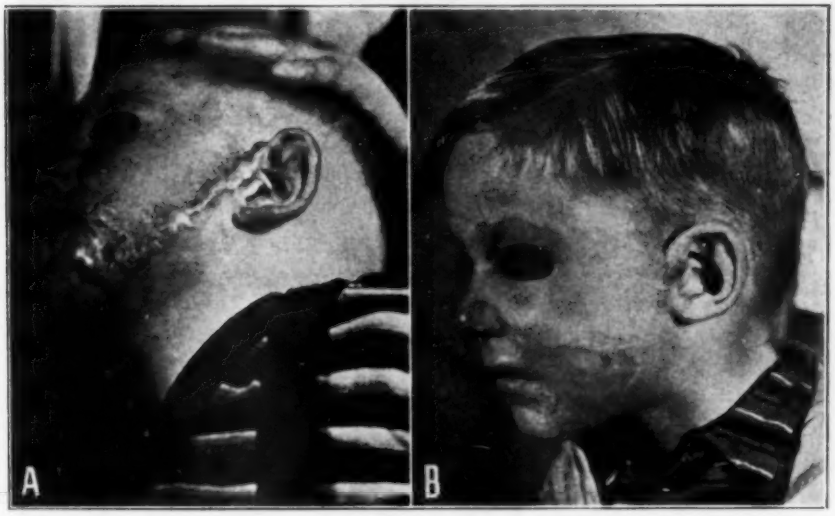

Fig. 5.—Hypertrophic scars of the face following third degree burns, treated by excision of scar and replacement with dermatome graft. Roentgen rays were given to the edges of the graft. *A*, appearance of the hypertrophic scar of the face prior to operation. *B*, appearance six months after replacement of the scar with a dermatome graft followed by roentgen therapy.

Fig. 6.—Hypertrophic scar involving the right shoulder, right side of neck and chin, right ear and right side of face, occurring in a poorly grafted third degree burn. *A*, appearance of the healed scar which at the time seemed to be developing into a progressive keloid. *B*, appearance after spontaneous regression of hypertrophy approximately a year later with residual scar contracture. *C*, good result from a large dermatome graft.

startling remedy to offer. We do wish to emphasize, however, that keloids and hypertrophic scars can and should be corrected or controlled by a judicious combination of surgical and roentgen therapy. Surgical excision alone cannot be expected always to solve the problem, for it is the tendency of these growths to recur in larger size after excision. It is for this reason that we employ roentgen ray therapy as an adjunct to operation in the treatment of keloids. It is not in our province to

Fig. 7.—Hypertrophic scars of the hands resulting from deep second degree burns caused by an explosion in a powder factory. These scars caused little or no impairment of function, but the patient was greatly distressed by the disfigurement. Appearance of the hands fifteen months after injury.

discuss this phase of the treatment except to mention that there is a variance of opinion as to how roentgen rays should be administered to these growths. Levitt and Gillies [5] maintained that single preoperative and postoperative treatments approximating a cutaneous erythema should

5. Levitt, W. M., and Gillies, H.: Radiotherapy in Prophylaxis and Treatment of Keloids, Lancet **1**:440-442 (April 11) 1942.

be given. Others [6] prefer a method of giving smaller divided doses beginning about three to five days after the excision.

Another measure which we wish to emphasize is the use of pressure in the control of hypertrophic scars and keloids. Nason, in reporting a comparison of four methods of treating keloidal growth, found results almost as good in treatment with pressure alone as he did with the combination of operation and roentgen ray therapy. Pressure may be simply applied by the repeated application of adhesive tape directly to the scar for a month or more following excision or by a more compli-

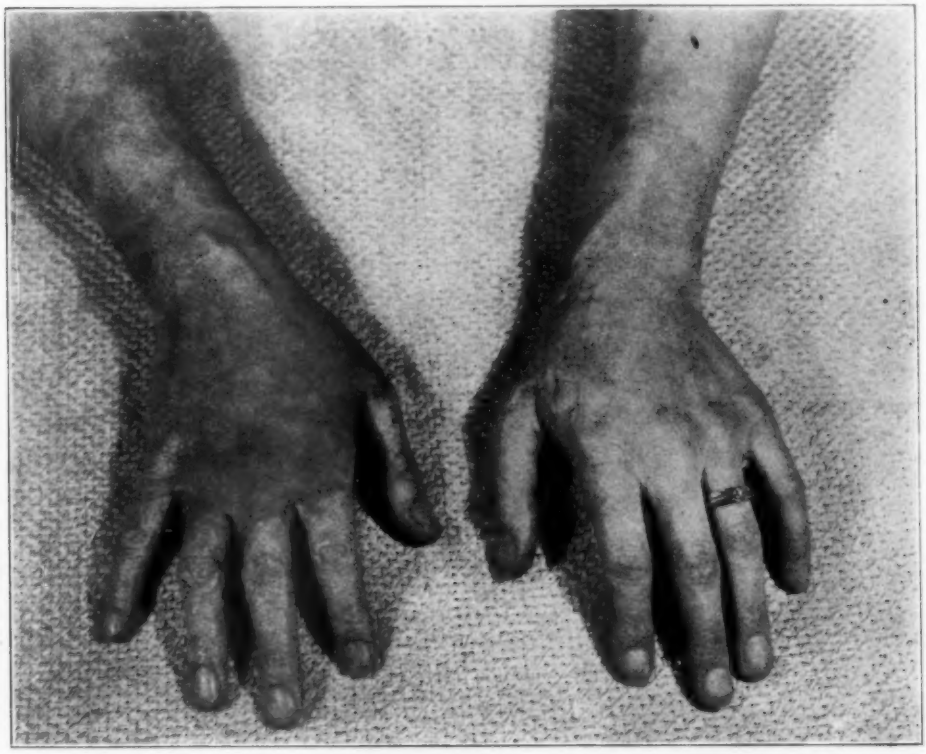

Fig. 8.—The same hands as shown in figure 7. Postoperative result after large split skin graft and roentgen therapy to the healed edges of the graft.

cated type of pressure dressing. Marshall and Rosenthal noted improvement in the appearance of keloids following the parenteral injection of a vasoconstricting material, a special fraction of liver extract. The theory behind this method is that vasoconstriction tends to reduce the

6. Allchin, F. M., cited by Matthews, D. N.: Surgery of Repair, Injuries and Burns, Springfield, Ill., Charles C Thomas, Publisher, 1947. Hunter, A. F.: Roentgen Therapy of Hypertrophic Scars and Keloids, Radiology **39**:400-409 (Oct.) 1942.

vascularity and decrease the size of the keloid by eliminating the tissue fluid. We personally have had no experience with this method of treatment. It would seem that pressure would assert its beneficial effect by directly eliminating this fluid and by reducing the vascularity.

At this point, we should like briefly to describe our own routine for the management of hypertrophic scars and keloids. Prevention rather than the later treatment is the thought uppermost in our minds. Since these growths tend to occur from three weeks to six months after operation, we attempt to keep the scars of all operative incisions and

Fig. 9.—Hypertrophic burn scars of the left arm and hand, showing little or no tendency to regress after several years. *A*, appearance of the deformed hand and arm prior to surgical intervention. *B*, appearance after four operations excising the scar and covering the defect with large dermatome grafts.

also the scars of accidental wounds and burns under frequent observation for this period of time. Since it is impossible to predict the extent to which hypertrophy will progress, roentgen therapy is ordered at the first sign of this tendency. Roentgen therapy cannot be expected to correct the fully matured keloid. As further insurance against hypertrophy, all scars are covered with adhesive for a period of about one month postoperatively unless there is some evidence of cutaneous sensitivity. In that case some other method of applying pressure is devised.

When confronted with keloids or hypertrophic scars which have already formed, the type of treatment depends on the location, size and age of the scar. Simple hypertrophic scars of limited extent are excised. The surgical technic is important. Since keloids tend to form in response to trauma, excision and closure should be as meticulous and nontraumatizing as possible. Incisions around the growth are made with a very sharp scalpel. Complete hemostasis is obtained. Fine-pointed forceps are used on the skin edges as sparingly as possible. In the closure subcutaneous sutures are placed only where necessary to obliterate dead space. Skin edges are approximated in accurate fashion with closely placed simple sutures of fine braided silk. We have noted no advantage to more elaborate types of suturing such as the subcuticular stitch or other suture material, such as wire. It is important that these

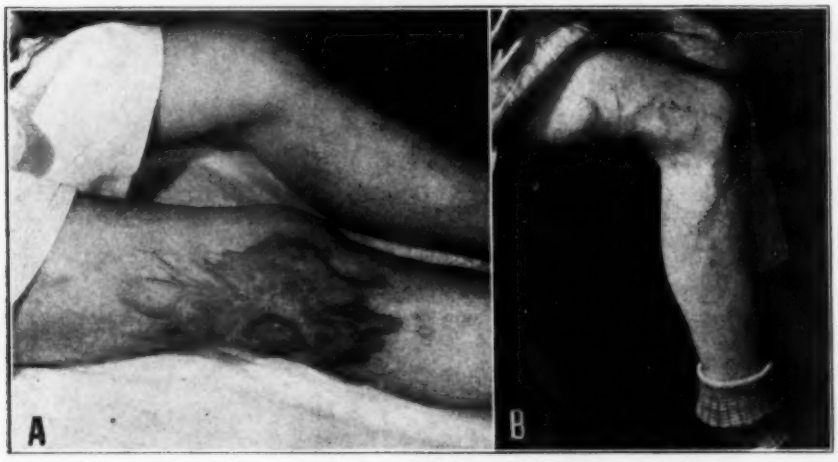

Fig. 10.—Hypertrophic burn scar with a tendency to progressive keloid formation due possibly to continued trauma and infection associated with chronic ulceration. *A*, preoperative appearance of the scar with central ulcer. *B*, result after complete excision and repair by split skin graft followed by roentgen therapy to the edges.

wounds not be closed under tension. If, after undermining the skin edges, tension still exists, relaxing incisions are made, provided that they can be placed in an area which will not interfere with the cosmetic result. Z-plastys are frequently employed to change the direction of scars that tend to cross the flexion creases. As soon as the sutures have been removed roentgen therapy should be given. For most cases we prefer the method of divided dosage. A 1 mm. aluminum filter being used, the first dose given [7] is ½ erythema dose. In two weeks this is repeated. For subsequent treatments the interval may be prolonged to

7. Collins, J. N.: Personal communication to the authors.

one month, unless the scar shows increase in size. The usual number of treatments is four.

Keloids which show signs of continuing activity when first seen are given a course of roentgen therapy prior to any surgical intervention.

Fig. 11.—Hypertrophic burn scar of the chest and abdominal wall and right axilla in a young girl. Appearance of the deformity several months after healing, showing the pronounced deformity of the breasts. The scar at this time was manifesting a tendency toward progressive keloid formation.

Operation is deferred until evidence of activity has subsided. Frequently a keloid is too large for complete excision and closure. It may, however, be of insufficient size to justify excision and skin graft. In this case,

the keloid is removed by a process of fractional excision, keeping entirely within the borders of the keloid and following each excision with roentgen therapy. Keloids which involve larger areas are completely excised and replaced by a large dermatome graft of intermediate thickness.

Fig. 12.—Result following surgical and roentgen treatment of the patient shown in figure 11. At the time of operation the axillary scar received subtotal excision and closure. The thoracic scar was resected sufficiently to release both breasts, and defects were covered with split skin grafts. Regression of all scars was prompt after a short course of roentgen therapy.

As soon as the graft has taken satisfactorily, roentgen rays are administered to the edges if scar hypertrophy appears.

Keloids which continue to recur after excision and a course of roentgen therapy are not subjected to continuing courses of roentgen rays. If the result is not satisfactory, the excision is repeated and, after this repair, additional roentgen therapy may be given. We wish to emphasize the obvious fact that repeated dosage and indiscriminate use of roentgen therapy is to be condemned. The technic we have employed has not caused any postirradiation complications. By carefully adhering to the general principles and procedures outlined, we have been able to prevent the formation of keloid scars in our practice and to give satisfactory correction for keloids which are presented to us already formed.

SUMMARY

An unsightly scar is often a matter of concern to a patient who acquires such disfigurement. It seems obvious, therefore, that the prevention and treatment of unsatisfactory scars should be of concern to the surgeon. Keloid growths and scar hypertrophy are the commonest causes of scar disfigurement.

Under the microscope it is difficult to distinguish the simple hypertrophic scar from a progressive keloid. In susceptible persons trauma to the dermis is the exciting factor in keloid formation. Among the theories which have been advanced to explain this susceptibility are "hormonal stimulation" and "fibroblastic diathesis," fibroblastic response to tissue edema occurring at the site of injury. Other factors which may influence keloid formation are cutaneous wounds which cross flexion creases, wounds which heal under tension or broad defects in which healing is delayed. The factors difficult to explain are the regional susceptibility to keloids and the manifestations of racial susceptibility and heredity.

Because of the fact that it is impossible to prognosticate the extent to which scar hypertrophy may progress it is our practice to keep all scars under close observation for at least three months after healing. We believe that keloids and hypertrophic scars can be prevented in a large number of cases by the continued application of pressure to the recently healed scar. If hypertrophy appears and cannot be controlled by pressure, roentgen therapy is instituted. Keloids and hypertrophic scars which have already formed are successfully treated by a combination of surgical excision, roentgen rays and pressure to the scar during the period of hyperplasia.

The surgical procedure for the correction of a keloidal scar must be selected according to the size and location of the lesion. Many conditions are best treated by fractional excision, keeping within the borders of the keloid and following each excision with roentgen therapy. The technic must be such that no permanent tissue damage results from the irradiation.

We wish to emphasize the importance of successful skin grafting in the prevention and treatment of keloids and hypertrophic scars.

DISCUSSION

Dr. Herman E. Pearse, Rochester, N. Y.: It was my privilege to spend last summer in Japan as a surgical consultant for the Atomic Bomb Casualties Committee. Four of us there were charged with the responsibility of evaluating cases of atomic bomb casualties, to decide the need for a long term study on the results of injuries. This necessitated visiting most of the Imperial universities of Japan, Hiroshima and Nagasaki. I had the opportunity of screening approximately 6,000 of the casualties and examining over 1,000 in order to evaluate their surgical lesions. It was my impression that, although the radioactivity of the atomic bomb might be lethal, the effects of the flash burn extended to a greater distance and gave deformities and disabilities of a long term nature. The greatest evidence of this disability was in the formation of keloids or hypertrophic scars. There is much to be learned about keloids. The impression exists that there is a growth potential, like that of a benign tumor, in the excessive formation of collagen. Any method of treatment which interferes with the blood supply may cause the keloids to regress, i. e., undercutting irradiation therapy or carbon dioxide snow. We have not the slightest conception of the basic causation of these lesions, and perhaps, if world conditions persist in being unsettled, it will behoove all of us to gain an interest not only in keloids but also in flash burns.

PARENTERAL USE OF WATER, DEXTROSE, ELECTROLYTES, FAT AND VITAMINS

WALTER G. MADDOCK, M.D.
CHICAGO

THIS ARTICLE is part of a symposium on "Nutritional Problems in Surgical Patients" and is a consideration of some therapeutic needs of persons who are unable or not permitted, for special reasons, to take sufficient fluids and food by mouth. Required water, dextrose, electrolytes and vitamins are then usually given parenterally, and present knowledge is reviewed with the object of aiding in the selection of the amount and kind of substances to administer. Fat is a relatively new material under experimentation for parenteral therapy, and its use will be briefly discussed.

Water and electrolyte balance is a complex matter in many ways and important disturbances in chemical concentrations occur in surgical and medical patients. But is is necessary to remember that in the great majority of cases treatment is and can be kept relatively simple. The instances of unusual and rather complicated chemical alterations require expert consideration and often special solutions, but their handling is not beyond the ability of the modern surgeon with a good background in the basic sciences. By citing the unusual exceptions, however, there is a tendency at this time to complicate the subject beyond practical use. There is also an inclination to depreciate the value of laboratory data. Chemical studies are extremely important and have furnished unlimited amounts of valuable information. Such data need to be interpreted with other laboratory data, such as urine and blood examinations, and with the history and physical findings of the patient.

From special studies, rules for the administration of water and electrolytes have been proposed, but the term "rule" is rather inflexible and some exceptions are usually found to them. "Working approximates" or "estimates" are better terms for the initial calculation of how much and what kind of fluid to give in a particular instance, and from that point modifications are made according to the response of the patient and further laboratory studies.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 21, 1948.

From the Department of Surgery of Wesley Memorial Hospital and Northwestern University Medical School, Chicago.

Whenever possible, information on water and electrolytes should be of a usable nature and should be presented to physicians in a manner to aid them in the care of their patients, and in the development of present information it is fitting to pay tribute to the important observations of O'Shaughnessy,[1] Schmidt,[2] Fagge,[3] Stadelmann,[4] Rogers,[5] Nichols and Andrews,[6] Trout,[7] Hartwell and Hoguet,[8] Henderson,[9] Macallum,[10] Matas,[11] Gamble,[12] Orr and Haden,[13] Van Slyke,[14] Peters,[15] Hartmann,[16] McCance,[17] Newburgh,[18] Coller,[19] Darrow,[20] Butler and Talbot.[21]

---

1. O'Shaughnessy, W. B.: Letter, London M. Gaz. **9**:486, 1831-1832.

2. Schmidt, K.: Characteristik der epidemischen Cholera gegenüber verwandten Transsudationsanomalieen, Leipzig, G. A. Reyher, 1850.

3. Fagge, C. H.: A Case of Diabetic Coma Treated with Partial Success by the Injection of a Saline Solution in the Blood, Guy's Hosp. Rep. **19**:173, 1874.

4. Stadelmann, E.: Ueber die Ursache der pathologische Ammonia-Kauscheidung beim Diabetes mellitus and des Coma diabeticum, Arch. f. exper. Path. u. Pharmakol. **17**:419, 1883.

5. Rogers, L.: The Variation in the Pressure and Composition of the Blood in Cholera and Their Bearing on the Success of Hypertonic Saline Transfusion in Its Treatment, Proc. Roy. Soc., London, **81**:219, 1909.

6. Nichols, H. J., and Andrews, V. L.: The Treatment of Asiatic Cholera During the Recent Epidemic, Philippine J. Sc., B. **4**:81, 1909.

7. Trout, H. H.: Proctoclysis: Some Clinical and Experimental Observations, J. A. M. A. **58**:1352 (May 4) 1912.

8. Hartwell, J. A., and Hoguet, J. P.: Experimental Intestinal Obstruction in Dogs with Especial Reference to the Cause of Death and the Treatment by Large Amounts of Normal Saline Solution, J. A. M. A. **59**:82 (July 13) 1912.

9. Henderson, L. J.: The Fitness of the Environment, New York, The Macmillan Company, 1930.

10. Macallum, A. B.: The Palaeochemistry of the Body Fluids and Tissues, Physiol. Rev. **6**:316 (April 26) 1926.

11. Matas, R.: The Continuous Intravenous "Drip," with Remarks on Value of Continued Gastric Drainage and Irrigation by Nasal Intubation with Gastroduodenal Tube (Gutte) in Surgical Practice, Ann. Surg. **79**:643 (May 24) 1924.

12. Gamble, J. L.: Dehydration, New England J. Med. **201**:909 (Nov. 7) 1929.

13. Orr, T. G., and Haden, R. L.; The Treatment of Intestinal Obstruction, Ann. Surg. **89**:354 (March 29) 1929.

14. Van Slyke, D. D.: Disorders of Metabolism: Acidosis and Alkalosis, Bull. New York Acad. Med. **10**:103 (March) 1934.

15. Peters, J. P.: Body Water: The Exchange of Fluids in Man, Springfield, Ill., Charles C Thomas, Publisher, 1935.

16. Hartmann, A. F.: Theory and Practice of Parenteral Fluid Administration, J. A. M. A. **103**:1349 (Nov. 3) 1934.

17. McCance, R. A.: Medical Problems in Mineral Metabolism: I. Legacies of Evolution, Lancet **1**:643 (March 21) 1936; II. Sodium Deficiencies in Clinical Medicine, ibid. **1**:704 (March 28) 1936.

18. Newburgh, L. H.; Johnston, M. W., and Newburgh, J. D.: Some Fundamental Principles of Metabolism, ed. 3, Ann Arbor, Mich., J. W. Edwards, 1948.

(Footnotes continued on next page)

For those wishing to review basic material on water and electrolyte balance I highly recommend Gamble's lecture syllabus on the chemical anatomy, physiology and pathology of extracellular fluid.[22]

WATER AND DEXTROSE

Water comprises about 70 per cent of the total body weight [23] and is distributed in two physiologic compartments, the intracellular and the extracellular. The intracellular fluid makes up approximately 50 per cent of the total body weight and the extracellular about 20 per cent. Water moves freely between these two compartments in response to osmotic forces. The effective osmotic pressure in the intracellular compartment is largely due to potassium, while in the extracellular fluid sodium takes its place, and the distribution of these two bases is the chief factor controlling the distribution of body water.[20]

The components of normal daily water exchange with some average figures are shown in table 1. As can be seen, water becomes available

TABLE 1.—*Components of Normal Water Exchange*

Available Water	Cubic Centimeters	Excretory Water	Cubic Centimeters
Fluids drunk	1,200	Water vaporized	1,200
Water from food		Water of stool	100
(a) Water content	1,000	Water of urine	1,200
(b) Water of oxidation	300		
Total	2,500		2,500

from fluids drunk and from food eaten, the latter providing both its water content and water of oxidation as it is burned for energy. In this way the ordinary solid foods of the diet, as meat, vegetables and fruit, yield about nine tenths of their weight as water and furnish an important part of the daily total.[24]

On the excretory side the normal water loss in the stool is insignificant, but two important processes require considerable daily volumes.

19. Coller, F. A.; Iob, L. V.; Vaughan, H. H.; Kalder, N. B., and Moyer, C. A.: Translocation of Fluid Produced by the Intravenous Administration of Isotonic Salt Solutions in Man Postoperatively, Ann. Surg. **122**:663 (Oct.) 1945.

20. Darrow, D. C.: The Treatment of Dehydration, Acidosis and Alkalosis, J. A. M. A. **114**:665 (Feb. 24) 1940.

21. Butler, A. M., and Talbot, N. B.: Parenteral Fluid Therapy, New England J. Med. **231**:585 (Oct.) 1944.

22. Gamble, J. L.: Chemical Anatomy, Physiology and Pathology of Extracellular Fluid, Cambridge, Mass., Harvard University Press, 1947.

23. Newburgh, Johnston and Newburgh.[18] Darrow.[20] Gamble.[22]

24. Coller, F. A., and Maddock, W. G.: Water Requirements of Surgical Patients, Ann. Surg. **98**:952 (Nov.) 1933.

Water is vaporized from the skin and lungs as part of the heat-dissipating mechanism, and without visible sweating average sized adults will use 1,000 to 1,500 cc. daily for this purpose.[25] Since an increase in the vaporization of water from the cutaneous surface is the safety valve of heat dissipation, hot humid environments may result in the use of up to 5 liters per day by this process. It is important to remember that the vaporization of water goes on even when available water is low in amount.

The kidneys use water to excrete waste materials in solution and do so with the amount available after the vaporization loss has been cared for. In this way the urine output is in most cases a good index of a satisfactory water balance.

ESTIMATION OF DAILY WATER REQUIREMENTS

There is no great reserve of water in the body. Therefore to prevent a rapid depletion in patients taking nothing by mouth a knowledge of the water needed for twenty-four hour periods is fundamentally important. With no ingestion of water or food, about 300 to 500 cc. of water becomes available from the water content and water of oxidation of an adult's own protein, fat and carbohydrate burned for energy.[26] It is better not to count on this source but to provide the patient with sufficient water to cover the two continuous daily losses, i. e., the water spent for vaporization and for urine.

From special studies the vaporization loss can be estimated as follows:[24] For the normal adult patient in a comfortable environment, allow 1,000 to 1,500 cc. per day. For patients having an extra load on the heat dissipating mechanism, as by fever, hyperthyroidism or a hot humid environment, allow 2,000 cc. for this process. For those who are perspiring most of the day much more water may be so lost, and figures up to 5,000 cc. have been obtained in the uncomfortably hot weather conditions.[27] By noting the presence and degree of sweating the careful observer will have no difficulty in allowing more water for the vaporization process.

Water for urine is obtained by giving an amount in excess of that used by the vaporizing process, and the important question is how much to allow for a satisfactory urine output. Lashmet and New-

---

25. Newburgh, L. H.; Wiley, F. H., and Lashmet, F. H.: A Method for the Determination of Heat Production over Long Periods of Time, J. Clin. Investigation **10**:703 (Oct.) 1931.

26. Newburgh, Johnston and Newburgh.[18] Gamble.[22]

27. Wassel, G. K.: Increased Vaporization of Water by the Postoperative Patient in Summer, Univ. Michigan Hosp. Bull. **4**:26 (April) 1938.

burgh [28] have furnished pertinent data in their determination of the minimal amount of water needed by persons with normal and with diseased kidneys to excrete a fixed amount of body wastes. From their work a calculation was made of the minimal amount of water needed to excrete 35 Gm. of waste material, which is about an average daily amount in health. From this calculation, shown in table 2, it is apparent that around 500 cc. of urine is the minimum for daily renal function. This figure is corroborated by Gamble,[22] who found 500 cc. of urine to be the obligatory outgo of healthy young men without food or water on life raft experiments.

This 500 cc., however, is too low an output of urine to be satisfactory for patients for many reasons. It occurs when the kidneys are working at maximum concentrating ability, as shown by a urine specific gravity of about 1.030. Since most body functions are not maintained at top speed, more water is needed to allow for renal function at moderate

TABLE 2.—*Minimal Amount of Water Needed to Excrete 35 Gm. of Waste Material* *

Status of Kidneys	Maximum Concentrating Ability, Specific Gravity	Minimum Water Needed, Cc.
Normal	1.032 to 1.029	483
Diseased†	1.028 to 1.025	595
	1.024 to 1.020	605
	1.019 to 1.015	850
	1.014 to 1.010	1,439

* Calculated from Lashmet and Newburgh.[28]
† Chronic nephritis, pyelonephritis, renal tuberculosis and other diseases.

working capacities, which means moderate urine specific gravity. Also, the sick patient is likely to have more than the average amount of waste materials to excrete, such as a greater amount of nitrogen from an increased breakdown of protein and/or ketone acid formation. A not infrequent additional difficulty in sick patients is a temporary or permanent decrease in the concentrating ability of the kidneys.

A further view of the inadequacy of low urine outputs comes from experimental studies and clinical observations on dehydration. Some adults with daily urine volumes close to 500 cc. are actually dehydrating. For example,[29] during a four day dehydration period a healthy medical student, J. N., on a very low water content diet and receiving no fluids for three days and only 514 cc. of water the fourth day was able to

28. Lashmet, F. H., and Newburgh, L. H.: A Comparative Study of the Excretion of Water and Solids by Normal and Abnormal Kidneys, J. Clin. Investigation **11**:1003 (Sept.) 1932.

29. Coller, F. A., and Maddock, W. G.: A Study of Dehydration in Humans, Ann. Surg. **102**:947 (Nov.) 1935.

excrete successive daily urine volumes of 472 cc., specific gravity 1.031; 481 cc., specific gravity 1.032; 442 cc., specific gravity 1.037, and 446 cc., specific gravity 1.038. During this period he lost 4,053 Gm. of body water, his blood nonprotein nitrogen increased from 31.0 to 45.7 mg. per hundred cubic centimeters and all of the common symptoms and signs of serious dehydration were present. From this example the seriousness of the situation when a patient excretes only 200 to 300 cc. of urine a day is apparent.

A satisfactory urinary output for the majority of surgical patients is 1,000 to 1,500 cc. per day.[30] As can be seen from table 2, it is two to three times the minimum and will allow for a moderate specific gravity in most instances. Other statements of a satisfactory daily urine volume are Graham, 900 cc.[31]; Butler and Talbot, 800 to 1,000 cc.[21]; Wangensteen, 1,000 to 1,500 cc.,[32] and Blalock, 1,500 cc.[33]

TABLE 3.—*Daily Water Requirements of Surgical Patients*

	Cubic Centimeters
Simple Case	
Water for vaporization	1,000 to 1,500
Water for urine	1,000 to 1,500
Total	2,000 to 3,000
Complicated Cases	
Water for vaporization (with sweating, fever, hyperthyroidism or hot humid environment)	2,000+
Water for urine	1,500+
Total	3,500+

More attention than is customary should be paid to the specific gravity of the urine. A specimen with a specific gravity close to 1.030 denotes excellent renal function and conversely an output of low fixed gravity comes from poor function. Table 2 shows that increasingly greater amounts of urine are needed to do the work as the specific gravity becomes less. With volumes of 1,500 to 3,000 cc. daily the patient with poor concentrating ability may be able to get rid of his daily waste materials and keep out of trouble. Similarly, persons with a retention of nitrogenous wastes or excess of electrolytes and organic acids may need 1,500 to 3,000 cc. of urine daily to correct these abnormalities.

30. Coller, F. A., and Maddock, W. G.: Water and Electrolyte Balance, Surg., Gynec. & Obst. **70**:340 (Feb.) 1940.

31. Graham, R. R.: Technical Surgical Procedures for Gastric and Duodenal Ulcer, Surg., Gynec. & Obst. **66**:269 (Feb. No. 2 A) 1938.

32. Wangensteen, O. H.: Care of Patient Before and After Operation, New England J. Med. **236**:121 (Jan. 23) 1947.

33. Blalock, A.: Principles of Surgical Care, Shock and Other Problems, St. Louis, C. V. Mosby Company, 1940.

In general the daily water requirements of surgical patients can be summarized [30] as in table 3. For the special circumstances discussed in previous and later paragraphs larger volumes may be needed.

KIND OF FLUID FOR DAILY WATER REQUIREMENTS

It should be clearly understood that the kind of parenterally administered fluid needed to provide water for vaporization and for urine is not sodium chloride solution, but water, with dextrose and/or a protein hydrolysate added for nutritional needs.

Dextrose Solutions, 5, 10 or 15 per Cent in Water.—These solutions are important materials for the patient taking insufficient water or food by mouth. The 5 and 10 per cent concentrations are most commonly used, and they provide water which appears to be available for all purposes, just as if it were taken by mouth,[34] and the carbohydrate

TABLE 4.—*Utilization of Five per Cent and Ten per Cent Dextrose Solutions by Surgical Patients* *

Factor	3,000 Cc. of 5% Dextrose in Water (150 Gm. Dextrose)	3,000 Cc. of 10% Dextrose in Water (300 Gm. Dextrose)
Patients showing glycosuria	80%	100%
Average amount dextrose retained	98%	95%
Average caloric value of dextrose retained	592 cal.	1,120 cal.
Dehydration effect	None	None
Irritation of veins	0-+	++

* From Winslow.[34b]

is burned for energy. It has been shown in volunteer subjects deprived of water, food and salt that 100 Gm. of carbohydrate daily decreases the rapidity of dehydration.[22] This excellent effect is considered to be due in part to water produced by the oxidation of the carbohydrate, to the reduced catabolism of protein and possibly to the avoidance of ketone acid formation, which may well play an important role in dehydration.

Most solutions for intravenous use contain at least 5 per cent dextrose, and a total of 2,000 to 3,000 cc. of such solution per day provides 100 to 150 Gm. of carbohydrate. The 400 to 600 calories so furnished is far below daily caloric needs, and the 10 per cent dextrose would be used oftener if it were not for its somewhat greater tendency to irritate veins and produce phlebothrombosis. Winslow [34b] compared the administration effect of approximately 3,000 cc. of 5 per cent dextrose in distilled water with the same amount of 10 per cent dextrose

34. (*a*) Gamble.[22] (*b*) Winslow, S. B.: Dextrose Utilization in Surgical Patients, Surgery **4**:867 (Dec.) 1938.

solution in two groups of surgical patients. The rate of administration was approximately 300 to 500 cc. per hour. The results shown in table 4 demonstrate the advantages of the 10 per cent solution. More carbohydrate was retained, and the average heat value of 1,120 calories was a great improvement, yet only about half of the amount required for an average-sized patient resting quietly in bed.[35] For greater caloric value the 15 per cent solution may come into use, particularly for patients with severe hepatic damage and malnutrition. For extremely ill patients dextrose solutions are advantageous preoperatively, during the operation and postoperatively, since among other benefits the glycogen content of the liver can be increased[36] and dextrose is an excellent cardiac stimulant and food for the myocardium.

Dextrose solutions have no specific value in shock, but along with the properly indicated blood, plasma and electrolytes, dextrose should be given for its general supportive effect.

Dextrose solutions are of no value in replacing electrolyte losses. Excellent examples of circulatory failure and shock have been noted in instances where salt-containing fluids were being lost, as by vomiting or enterostomy drainage, and only dextrose solutions were being given intravenously. This is a direct contravention of the general principle that parenteral fluid therapy should provide as nearly as possible the kind of material lost.

Dextrose solutions supply easily available water for countless internal fluid adjustments and for the replacement of water used for vaporization and kidney function. A good supply of available water is of tremendous importance to the kidneys, since it enables them in health and aids them in disease to guard the proper concentration of substances in solution in the blood. This will be emphasized later.

Protein Hydrolysate Solutions.—In this symposium, protein hydrolysate solutions are being discussed by Dr. Robert Elman of St. Louis, who has made a great contribution in their development and use. These solutions provide available water and protein, and usually some carbohydrate because they are commonly made up with 5 per cent dextrose. Some of them also contain 2 Gm. of sodium chloride per liter and others do not, so that the preparation label should be read to learn exactly what is being given. I select a protein hydrolysate solution for its protein content, for patients who showed undernutrition before operation and for those not likely to begin eating soon after operation.

---

35. Newburgh, L. H.: The Importance of Actually Measuring the Total Heat Production, Ann. Int. Med. **8**:459 (Oct.) 1934.

36. MacIntyre, D. S.; Pedersen, S., and Maddock, W. G.: McClure-Aldrich Test in Water Balance, Surg., Gynec. & Obst. **72**:834 (May) 1941.

DAILY ELECTROLYTE REQUIREMENTS

The fact is that there are no data on hand to prove or disprove the thought that generally a few grams of sodium chloride should be given daily to patients taking nothing by mouth, having no abnormal electrolyte losses, but requiring nutritional substance parenterally. It is true that if such patients could take substance orally their normal taste desires would include some sodium chloride. It is not illogical then if sustenance depends on parenteral methods to give a small amount, about 5 Gm., of sodium chloride to these patients daily. Butler and Talbot [21] place the normal daily allowance of sodium chloride at 1 Gm. for infants, 3 Gm. for children and 6 Gm. for adolescents and adults. Conveniently for adults 500 cc. of isotonic sodium chloride solution or Ringer's solution daily will provide this need, or a liter can be given every second day.

As suggested by Coller and his co-workers in a study of salt intolerance in the immediate postoperative period,[37] sodium chloride should not be given for two days postoperatively unless specifically indicated by a considerable gastrointestinal fluid loss. Similarly, when the other factors of abnormal water and electrolyte retention are present—malnutrition, sepsis, severe hemorrhage, serum loss, as from burn surfaces, renal, hepatic or cardiac damage [38]—the daily electrolyte allowance may well be further reduced or dispensed with. Moyer [39] expressed the belief that the incidence of postoperative pulmonary complications after surgical treatment of the upper part of the abdominal tract was higher, especially in aged persons, when maintenance doses (5 to 9 Gm. daily) were infused than when no salt was given unless specifically indicated.

ELECTROLYTES AND DEHYDRATION

Patients who are dehydrated present a special problem in fluid balance because they have a fluid deficit. To make up for this deficit they need: (1) more than daily maintenace amounts of fluid and (2) water plus electrolytes. As discussed later a deficient water intake will produce dehydration, but the condition develops most commonly in patients who are losing digestive tract fluids as by vomiting and with continuous gastrointestinal aspiration, biliary drainage, pancreatic fistulas, ileostomy drainage and diarrhea. All such fluids contain important amounts of electrolytes, so that for practical purposes dehydration must be a consideration of water and electrolyte loss.

---

37. Coller, F. A.; Campbell, K. N.; Vaughan, H. H.; Iob, L. V., and Moyer, C. A.: Postoperative Salt Intolerance, Ann. Surg. **119**:533 (April) 1944.

38. Jones, C. M., and Eaton, F. B.: Postoperative Nutritional Edema, Arch. Surg. **27**:159 (July) 1933.

39. Moyer, C. A.: Fluid and Electrolyte Balance, Surg., Gynec. & Obst. **84**:586 (April) 1947.

Electrolytes are of great consequence to the body, for they form a most important part of the fluids that pervade all tissue and they are responsible for a large percentage of the total osmotic pressure of the body fluids. In the extracellular compartment which can be subdivided into the vascular and the interstitial spaces, sodium and chloride and bicarbonate ions are of greatest abundance, while potassium, calcium, magnesium, sulfates and phosphates are in lesser amounts. In dehydration the loss of fluid volume is greater in the extracellular compartment, though not entirely so, and considering that its fluid is the circulating medium that brings every kind of nourishment to the cells, takes away all waste materials, provides the digestive juices and furnishes water for heat dissipation and for urine, it is not surprising that an appreciable loss of this fluid produces profound effects.[20]

CHEMICAL STUDIES

The chemical studies usually made on patients with dehydration are the combining power of plasma carbon dioxide, the plasma or whole blood chlorides, the blood nonprotein or urea nitrogen and the plasma proteins. Lately, faster methods have made serum sodium studies possible, which is a real advance, and a renewed awareness of the importance of potassium has resulted in studies of blood potassium. Some pertinent comments on these laboratory data and their application follow.

Combining Power of Plasma Carbon Dioxide (Normal 55 to 65 Volumes per Cent).—This test is the simplest and most dependable measurement for the presence of alkalosis or acidosis [22] and is carried out by determining the volume of carbon dioxide released by adding strong acid to a known amount of plasma in the Van Slyke burette. Changes in the carbon dioxide volume from the normal may be due to two possible variables:

1. Carbonic acid ($H.HCO_3$), which is formed from the carbon dioxide of body metabolism; its normal plasma concentration depends on the respiratory mechanism removing carbon dioxide through the lungs. Important disturbances of respiratory control are relatively infrequent. Alkalosis due to carbonic acid deficit has been noted with overventilation of a voluntary nature, or with encephalitis or hysteria or to a mild degree in febrile states.[14] The acidosis of carbonic acid excess has been seen in some cases of emphysema, asthma and depression of breathing by opiates, in which gas exchange in the lungs is decidedly hindered.[14]

2. Base bicarbonate ($B.HCO_3$), which varies with electrolyte disturbances, such as produced by a loss of gastrointestinal tract fluids through vomiting, biliary and pancreatic fistulas, ileostomy drainage and diarrhea. Occasionally medication, such as large amounts of sodium

bicarbonate or sodium phosphate, disturbs electrolyte balance. Important changes also result from the retention of phosphoric and sulfuric acid in nephritis and ketone acids in diabetes and the quick ketosis of children. In these chemical disturbances alkalosis or alkali excess is indicated by a carbon dioxide combining power of 75 or 80 volumes per cent or more [14] and occurs with large losses of chlorides, as by the vomiting of pyloric obstruction or by the excessive or prolonged administration of sodium bicarbonate or phosphate. On the other hand, acidosis or alkali deficit of a significant degree is indicated by a carbon dioxide combining power of 40 volumes per cent or below [14] and most commonly occurs when large amounts of base bicarbonate are lost by diarrhea, biliary tract or ileostomy drainage and the retention of acids in nephritis and ketosis.

In the diagnosis of alkalosis or acidosis [40] a determination of the combining power of plasma carbon dioxide suffices when there is a history of gastrointestinal fluid loss, nephritis or diabetes. In such

TABLE 5.—*Correlation of Carbon Dioxide Combining Power with Blood p_H to Indicate the Chemical Disturbance*

Carbon Dioxide Combining Power	Blood p_H	Metabolic Disturbance (Common)	Respiratory Disturbance (Uncommon)
High	Above 7.4	Alkalosis	
	Below 7.4		Acidosis
Low	Below 7.4	Acidosis	
	Above 7.4		Alkalosis

"metabolic" disturbances, which make up the great majority of the clinical cases, a high combining power of carbon dioxide dependably indicates alkalosis and low value acidosis. When, as occasionally occurs, "respiratory" factors from any cause produce underventilation or overventilation, and thus alter concentrations of plasma carbonic acid, the changes in the combining power of carbon dioxide have the reverse significance: a high value indicates acidosis and a low value indicates alkalosis. Therefore, with respiratory factors the plasma p_H should be determined and correlated with the carbon dioxide combining power, as shown in table 5, to indicate the proper reaction disturbance. A recent case emphasized this need. Both metabolic and respiratory conditions were present, since an inlying Wangensteen tube was continuously removing gastrointestinal fluid and the respiratory rate was decidedly increased because of abdominal distention and pneumonitis.

Serum Sodium (Normal 326 Mg. per Hundred Cubic Centimeters, or 142 Milliequivalents per Liter).—Serum sodium values give direct information on the predominant basic ion in extracellular fluids and

40. Van Slyke.[14] Gamble.[22]

are important determinations in all body electrolyte studies. Unfortunately, the chemical analysis of a serum sodium value takes considerable time, so that this test has been used mainly in experimental studies and seldom in general clinical work. Recently flame photometer methods have been developed which take much less time,[41] and it is hoped that simple reliable equipment will soon be available to make this study possible in most hospital laboratories.

Serum Potassium (Normal 20 Mg. per Hundred Cubic Centimeters, or 5 Milliequivalents per Liter).—The importance of potassium in dehydration states has lately been reemphasized by Govan and Darrow,[42] who by its use with sodium chloride solutions reduced the mortality of infants with infectious diarrhea from 17 in 53 to 3 in 50. Potassium is, therefore, discussed later in this paper.

Plasma Chlorides (Normal 560 Mg. per Hundred Cubic Centimeters Expressed as Sodium Chloride and as Straight Chloride 365 mg. per Hundred Cubic Centimeters, or 103 Milliequivalents per Liter).—This determination is an important one, particularly in patients who have lost significant amounts of gastrointestinal tract fluids. There are some special aspects to remember about the data obtained:

1. Chloride loss receives much attention, possibly more than it merits when the concurrent loss of other substances is considered. When chloride is lost ionic equilibrium among the acid radicals of extracellular fluid is maintained by an increase in bicarbonate. Actually sodium loss is more important than chloride loss, but, relatively, sodium studies are harder to do and, therefore, chloride studies are made and receive more attention. In general a lowered level of plasma chloride is one indication of electrolyte loss, which means, importantly, an extracellular fluid loss.

2. A lowering of the level of plasma chlorides reflects a fall in the chloride level of other body fluids. The data are not always quantitative, but from work on dogs vomiting from intestinal obstruction White and Bridge [43] concluded that chlorine is lost from the body in amounts directly proportional to the decrease in chlorine content in the blood and the total dechlorination of the body corresponds closely to the salt

41. Hoffman, W. S., and Osgood, B.: The Value of Blood Chloride and Sodium Determinations in the Diagnosis of Dehydration, J. Lab. & Clin. Med. **27**:1174 (June) 1942.

42. Govan, C. D., Jr., and Darrow, D. C.: The Use of Potassium Chloride in the Treatment of the Dehydration of Diarrhea in Infants, J. Pediat. **28**:541 (May) 1946.

43. White, J. C., and Bridge, E. M.: Loss of Chloride and Water from the Tissues and Blood in Acute High Intestinal Obstruction, Boston M. & S. J. **196**: 893 (June 2) 1927.

recovered in the vomitus and urine. The observation of low levels of plasma chlorides in patients with a history of the abnormal loss of gastrointestinal tract fluid, particularly from vomiting or the aspiration of fluid from the upper part of the gastrointestinal tract, roughly reflects the extent of the loss.

3. A plasma chloride value is not a good indicator of an excessive administration of a sodium chloride solution to the seriously ill patient. The water and electrolytes administered to these patients pass quickly into the interstitial spaces and results generally in hyperhydration and edema rather than hyperchloremia.[44] It is unfortunate that in the sick patient the excessive administration of sodium chloride is not shown by plasma chloride levels rising rapidly above the normal, since if they did it would be simple to follow chloride administration to the normal by repeated tests.

4. Failure in a seriously ill patient to raise a low plasma chloride level with a sodium chloride solution, as previously mentioned, most commonly is associated with the sodium chloride solution's going to the interstitial spaces and producing hyperhydration and edema.[45] Occasionally a sick patient will excrete the salt in the urine even though the plasma concentration remains below normal. The reason for this is not entirely clear, but the best evidence is that the kidneys may have temporarily lost their ability to retain needed electrolytes. It has been definitely noted that if the general condition of these patients improves, often meaning that the plasma protein level improves,[46] a shift may occur in the electrolyte distribution and plasma chlorides increase to normal without further administration of sodium chloride.

5. While moderately or seriously ill, most patients do not maintain plasma chloride levels at the normal figure, but at a somewhat lower level. This has been noted in burns and other diseases when an

---

44. (*a*) Bartlett, R. M.; Bingham, D. L. C., and Pedersen, S.: Salt Balance in Surgical Patients, Surgery **4**:441 (Sept.); 614 (Oct.) 1938. (*b*) Maddock, W. G., and Coller, F. A.: Sodium Chloride Metabolism in Surgical Patients, Ann. Surg. **112**:520 (Oct.) 1940. (*c*) de Wesselow, O. L. V.: The Variations in the Chloride Content of the Blood, Internat. Clin. **3**:191 (Sept.) 1924.

45. (*a*) Power, F. H.; Pedersen, S., and Maddock, W. G.: Clinical Experiences with Sodium Chloride Replacement, Surgery **12**:438 (Sept.) 1942. (*b*) Coller, F. A.; Bartlett, R. M.; Bingham, D. L. C.; Maddock, W. G., and Pedersen, S.: The Replacement of Sodium Chloride in Surgical Patients, Ann. Surg. **108**:769 (Oct.) 1938.

46. Maddock and Coller.[44b] Ariel, I.; Abels, J. E.; Pack, G. T., and Rhoads, C. P.: Metabolic Studies of Patients with Cancer of the Gastro-Intestinal Tract: XVI. The Treatment of Hypochloremia Refractory to the Administration of Sodium Chloride, Especially in Patients with Gastrointestinal Cancer, J. A. M. A. **123**:28 (Sept. 4) 1943.

abnormal shift of water and electrolytes may have taken place.[30] There may be an excessive amount of edema fluid either generally or locally about an injured part, or excess fluid may have accumulated in the thoracic or abdominal cavities. A patient is probably not suffering from a lack of chlorides with a plasma level down to 500 mg. per hundred cubic centimeters, and often while the patient is seriously ill administration of sodium chloride will not raise the value to the normal level.[45] In these cases the history, physical observations, other laboratory data and the general condition of the patient should be considered.

One should be particularly careful about administering isotonic sodium chloride solutions to patients whose plasma chloride level is moderately low but who have no history of the abnormal loss of electrolytes, as by vomiting. For example, a patient with ascites is taking sodium chloride from other parts of the body to make the ascitic fluid, and his plasma chloride level is likely to be below the normal of 560 mg. per hundred cubic centimeters.[47] If a sodium chloride solution is given it may elevate the plasma electrolytes temporarily, but more ascitic fluid will be promptly formed and the plasma level will drop again.[44a]

6. A word about studies of whole blood chlorides. These values vary with the degree of anemia, and the test has been criticized for that reason. Hoffman and Osgood[41] recently emphasized the pronounced variations that occur with different hemoglobin concentrations or hematocrit readings and pointed out the need for a chart of these interrelations in interpreting values of whole blood chlorides. Their conclusion was that the test of whole blood chlorides should be abandoned and studies of plasma chlorides substituted.

Blood Nonprotein Nitrogen.—Evidence of retention of waste materials is commonly found with the oliguria of dehydration. Two factors are generally accepted as important in its production: (1) insufficient water for renal function and (2) increased destruction of protein associated with the disease. There may also be a temporary decrease in concentrating ability of the kidneys at this time, so that with a given amount of available water less waste materials are excreted.[29]

Plasma Proteins.—These are importantly concerned with water and electrolyte shifts in the body. With low levels of plasma proteins, there is a striking tendency for the abnormal retention of fluids and edema formation is common. As mentioned previously, many surgical patients are malnourished because of their disease, so that low levels of plasma

47. Bartlett, Bingham and Pedersen.[44a] Porges, O.: Ueber Coma hypochloraemicum, Klin. Wchnschr. **11**:186 (Jan. 30) 1932. Mach, R. S.: Les manifestations cliniques de l'hypochlorémie, Rev. méd. de la Suisse Rom. **54**:829 (Aug. 25) 1934.

proteins often confront the surgeon.[48] Retention of fluids should be thought of as occurring generally through interstitial spaces and locally at injury and operative sites. In the latter regard, Mecray, Barden and Ravdin [49] have described obstruction at gastroenterostomy stomas from edema associated with low levels of plasma proteins.

Moore and Van Slyke [50] have stated the following critical levels at which fluids tend to be retained: total plasma proteins 5.5 mg. and plasma albumin 2.5 mg. per hundred cubic centimeters. These are good figures to remember, but it has been noted by many workers [51] that sodium chloride solutions may be retained and edema may develop in the seriously ill patient when plasma proteins are normal or just slightly below normal.

SYMPTOMS AND SIGNS OF DEHYDRATION

A number of variables are important in the symptoms and signs of dehydration. The more rapid the deficit the more acute the symptoms, and, conversely, with slowly developing deficits there may be few symptoms. The poorer the general condition of the patient and the older the patient the severer the reaction.[39]

The manner in which the dehydration develops also makes a difference. In 1935 Kerpel-Fronius [52] demonstrated in rabbits two types of dehydration: (1) dehydration due to a shortage of water, but not accompanied with a corresponding loss of salt, and characterized by thirst and (2) dehydration due to a primary loss of electrolytes, which produces a more profound dehydration and is characterized by circulatory disturbances and collapse. McCance [17] and Nadal and his co-work-

48. (*a*) Jones and Eaton.[38] (*b*) Cantarow, A.: Water Balance; Edema and Dehydration, Internat. Clin. **1**:266 (March) 1939. (*c*) Coller, F. A.; Dick, V. S., and Maddock, W. G.: Maintenance of Normal Water Exchange with Intravenous Fluids, J. A. M. A. **107**:1522 (Nov. 7) 1936. (*d*) Curphey, W. C., and Orr, T. G.: Edema in Surgical Patients, Surgery **1**:589 (April) 1937. (*e*) Weech, A. A.; Snelling, C. E., and Goettsch, E.: The Relation Between Plasma Protein Content, Plasma Specific Gravity and Edema in Dogs Maintained on a Protein Inadequate Diet and in Dogs Rendered Edematous by Plasmapheresis, J. Clin. Investigation **12**:193 (Jan.) 1933.

49. Mecray, P. M., Jr.; Barden, R. P., and Ravdin, I. S.: Nutritional Edema: Its Effect on the Gastric Emptying Time Before and After Gastric Operations, Surgery **1**:53 (Jan.) 1937.

50. Moore, N. S., and Van Slyke, D. D.: The Relationships Between Plasma Specific Gravity, Plasma Protein Content and Edema in Nephritis, J. Clin. Investigation **8**:337 (April) 1930.

51. Coller, Dick and Maddock.[48e] Maddock, W. G., and Coller, F. A.: Water Balance in Surgery, J. A. M. A. **108**:1 (Jan. 2) 1937. Curphey and Orr.[48d] Power, Pedersen and Maddock.[45] Jones and Eaton.[38]

52. Kerpel-Fronius, Ö.: Ueber die Beziehungen zwischen Salz- und Wasserhaushalt bei experimentellen Wasserverlusten, Ztschr. f. Kinderh. **57**:489, 1935.

ers [53] studied the type in experimental human subjects and again noted the different responses. With no or very little water intake there is considerable thirst and a dry mouth and tongue, oliguria with high specific gravity of the urine if renal function is good, and a slow increase in the nitrogenous wastes in the blood, hallucinations, disorientation and coma as the deficit becomes extreme. Plasma protein and hematocrit concentrations may not be significantly altered until very late.

The dehydration of electrolyte fluid loss with plenty of water intake is an experimental setup or an example in a patient of the thoughtless replacement of an electrolyte fluid loss by water and some dextrose. With a good water intake such patients are not thirsty.

Most clinical instances of dehydration from electrolyte fluid loss are combined with water deficit, since the patient who is vomiting is usually not drinking. The result is an extracellular fluid loss. When it is slight or of moderate degree there is some apathy, weakness, somnolence, anorexia, nausea and variable changes in systolic blood pressure. With severe dehydration these signs and symptoms are increased, until finally circulatory collapse with blood pressure down to shock levels appear. The skin and tongue are dry and wrinkled, the muscles feel puttylike, the eyeballs are soft and the body temperature may be subnormal. As the dehydration progresses the hematocrit and plasma protein concentrations usually increase while the electrolytes decrease. Urinary output is usually rather low, and blood urea or nonprotein nitrogen levels are increased. All these manifestations are severer with rapidly developing deficits.

To be alert to recognize dehydration is to be aware of the possibilities and to look for them. The chief candidates are patients losing important amounts of gastrointestinal fluids, or those with the chemical disturbances of diabetic coma. Nondiabetic ketosis, nephritis, burns, Addison's disease, heat prostration and heat cramps are other causes not infrequently seen.[20] The presence of the severe symptoms and signs of dehydration so often indicate the failure to make an early diagnosis and to employ proper treatment.

SPECIFIC EXAMPLES OF WATER AND ELECTROLYTE LOSS

As just stated, important losses of water and electrolytes in surgical patients occur mainly with the abnormal loss of gastrointestinal tract fluids, and vomiting or the continuous aspiration of fluid to prevent vomiting is the commonest cause. Other serious water and electrolyte

53. Nadal, J. W.; Pedersen, S., and Maddock, W. G.: A Comparison Between Dehydration from Salt Loss and from Water Deprivation, J. Clin. Investigation **20**:691 (Nov.) 1941.

alterations mentioned are those occurring with ketosis, with disturbances of renal function in nephritis and in burn patients. These situations will be discussed briefly.[54]

In considering gastrointestinal fluid losses the difference in the chemical pattern of the juices in different parts of the tract must be remembered, because their loss produces different effects. Therefore, the concentration of the chief electrolytes in the important digestive fluids is shown in table 6.

TABLE 6.—*Concentration of Electrolytes in Some Digestive Fluids and in Commonly Used Restorative Solutions*

Source	Milliequivalents per Liter			Sodium Chloride, Gm./L.	
	Sodium	Chloride	Bicarbonate	Range	Average
Fundus gastric juice*	27	158	..	.....	...
Pylorus gastric juice*	157	142	..	.....	...
Total gastric juice†	22	145	..	.....	...
Vomitus‡	3-78	20-106	..	1.2-5.5	...
Vomitus§	...	...	..	1.2-6.2	3.3
Duodenum*	165	118	..	.....	...
Gastroduodenal drainage§	...	...	..	1.9-7.9	5.7
Hepatic duct bile†	142	108	40	.....	...
Hepatic duct bile‡	147	130	..	.....	...
Hepatic duct bile§	...	...	..	3.5-6.4	5.1
Pancreatic juice*	157	115	43	.....	...
Pancreatic juice†	145	38	113	.....	...
Jejunal secretion†	141	115	32	.....	...
Enterostomy drainage‡	95-106	74-103	..	.....	...
Intestinal fistula drainage§	...	...	..	3.0-8.8	5.2
Ileal secretion*	155	81	90	.....	...
Colon secretion*	153	83	91	.....	...
Diarrheal stool§	...	...	..	3.7-5.2	4.3
Isotonic sodium chloride solution	155	155	..	.....	9.0
Ringer's solution	148	158	..	.....	8.6
Potassium 4				Potassium chloride	0.3
Calcium... 6				Calcium chloride..	0.33
Lactated Ringer's	125	111	..	Sodium chloride....	6.0
Potassium 4				Sodium lactate.....	2.5
Calcium... 4			Lactate 22	Potassium chloride	0.3
				Calcium chloride...	0.2

* Peters and Van Slyke.[54b]
† Gamble.[22]
‡ Dick, Maddock and Coller.[75a]
§ Bartlett, Bingham and Pedersen.[44a]

Vomiting From Pyloric Obstruction.—In infants the common cause of such an obstruction is congenital hypertrophic pyloric stenosis, while in adults the lesion is usually carcinoma of the stomach or scar contraction from a long-standing duodenal ulcer. Without achlorhydria, the vomitus is predominantly acid and the result is a lowered value of plasma chloride and a relative increase in plasma base as shown by an increase combining power of carbon dioxide. Actually there is some loss of sodium, but it is so much less than the loss of chlorides that

54. (*a*) Van Slyke.[14] Darrow.[20] Butler and Talbot.[21] Gamble.[22] Coller and Maddock.[80] Moyer.[39] (*b*) Peters, J. P., and Van Slyke, D. D.: Quantitative Clinical Chemistry: Baltimore, Williams & Wilkins Company, 1931, vol. 1.

the total base is relatively increased. In cases of severe disease the picture is typically that of alkalosis, and while tetany from it is rare, dehydration is common. Most workers agree that isotonic sodium chloride solution or Ringer's solution is suitable for the replacement therapy in the great majority of cases. Only in severe alkalosis is hydrochloric acid or ammonium chloride considered.

Vomiting From Duodenal, Jejunal or Upper Ileal Obstruction.—The regurgitation of digestive fluid from these levels brings back a combination of saliva, gastric juice, bile, pancreatic juice and succus entericus. Some of these fluids have more chloride ions than basic ions, while others have the reverse (table 6). The sum is a loss of more nearly equal amounts of acid and base, so that chemical studies of such patients usually show a definitely lowered value of plasma chloride and a variable combining power of carbon dioxide, generally not far from normal. The latter test, because it is relative to the strong acids, such as chloride, at the time of the test, indicates a definitely lowered level of serum base. If studies of serum sodium are made the results will be found definitely below normal. In these cases isotonic sodium chloride solution or Ringer's solution is a suitable replacement fluid.

Bile or Pancreatic Fistula Drainage.—Drainage of the common bile duct is a frequent surgical procedure which often results in a daily loss of from 500 to 2,000 cc. of bile. As can be seen from table 6, the base concentration of bile is greater than the chloride, and as a result inorganic acidosis can develop. Pancreatic fluid has a somewhat similar acid-base pattern, and its loss will produce the same result. In serious dehydration from these causes, the plasma chlorides are usually only moderately reduced while the combining power of carbon dioxide or the level of serum sodium will be lowered to acidotic levels. Corrective fluid can be isotonic sodium chloride solution or Ringer's solution, but one of the so-called balanced solutions, such as lactated Ringer's solution U. S. P., is better because it provides the needed excess of sodium.

Ileostomy Drainage and Diarrhea.—The effect of loss of material from these two sites is about the same as loss of bile or pancreatic juice. In each instance the sodium loss is greater than the chloride loss, so that with a copious drainage close attention must be paid to the electrolytes or a severe inorganic acidosis will develop. The level of plasma chlorides will be slightly or moderately lowered, while the combining power of carbon dioxide or serum sodium will be well down. Some of these patients, particularly the adults with more chronic illness, can eat and can thus take sodium bicarbonate and sodium chloride tablets by mouth. Thus it is not always necessary to give electrolytes parenterally to maintain normal levels in such patients.

As with severe diarrhea, ileostomy drainage has been associated with severe extracellular fluid depletion and shock.[55] It is somewhat of a surprise to see extreme malaise, apathy, stupor and shock with very low levels of blood pressure respond promptly to the administration of an electrolyte solution. It is an example again of the proper replacement principle, that is, of giving the patient as nearly as possible what he has lost.

The best replacement solution if parenteral administration is necessary in cases of ileostomy drainage or diarrhea is lactated Ringer's solution, since it provides the needed excess of sodium. In some instances of severe acidosis sodium bicarbonate is given. Its use will be discussed later.

Ketosis of Diabetic and Nondiabetic Origin.[56]—In diabetes, ketosis develops from a lowering of carbohydrate metabolism to the point where incompletely oxidized fatty acids begin to appear in extracellular fluid. Also, in many disease conditions partial or complete starvation reduces carbohydrate metabolism and ketosis develops. The kidneys respond by excreting the ketone acids in the form of their sodium and ammonium salt. With ketosis severe dehydration is common, and as a sequela there is some loss of renal control, permitting the abnormal retention of sulfuric and phosphoric acid radicals and the excretion of sodium chloride. A further depletion of electrolytes may occur from the vomiting so commonly associated with ketone-producing diseases. Treatment requirements are met by large amounts of lactated Ringer's or isotonic sodium chloride solution. If the latter is used an excess of sodium is often given by the addition of a sodium bicarbonate or sodium lactate solution. The intravenous administration of dextrose solutions is also needed to overcome ketosis, with the addition of insulin if it is of diabetic origin.

Nephritis.[56]—In both acute and chronic nephritis the development of acidosis is not uncommon. Altered renal function may result in retention of sulfuric and phosphoric acid radicals, and at the same time a loss of sodium and chloride ions occurs from a failure to reabsorb them from the glomerular filtrate back to the plasma. A further serious disturbance in severe nephritis is a diminution or loss of ammonia formation in the kidneys. This process ordinarily allows the excretion of abnormal acids as their ammonium salt and thus spares the alkali reserve, but it fails sometimes in nephritis. Thus, dehydration

55. Cave, H. W.: Late Results in the Treatment of Ulcerative Colitis, Ann. Surg. **124**:716 (Oct.) 1946. Welch, C. S.; Masson, J. C., and Wakefield, E. G.: Clinical and Laboratory Findings After Excessive Loss of Intestinal Fluid from the Ileum, Surg., Gynec. & Obst. **64**:617 (March) 1937.

56. Van Slyke.[14] Newburgh, Johnston and Newburgh.[18] Darrow.[20] Gamble.[22]

with deficiency of extracellular electrolytes of an acidotic pattern is not uncommon. Darrow [20] stated that although the patients may be edematous the serum concentrations of sodium are usually low, and small amounts of interstitial salt solution [57] intravenously or of sodium chloride and sodium bicarbonate by mouth, enough to replace the urinary loss, are often beneficial to increase the diuretic effect of the good supply of water also given.

Burns.[58]—For twenty-four to thirty-six hours after burns there is an outpouring of plasma into the skin and subcutaneous tissue of the burned area that causes a decrease in circulating plasma volume and a hemoconcentration roughly proportional to the depth and extent of the burn. Electrolytes as part of the plasma fluid are trapped in the burned area, and their concentration in the circulating plasma is mildly to moderately decreased. With no actual loss from the body at the time of the burn Maddock and Coller [44b] considered that no more than moderate amounts of electrolyte solutions intravenously should be given to burned patients, because with their usual pronounced acute hypoproteinemia salt solution further reduced the plasma protein concentration and produced edema. Darrow,[20] however, stated the belief that isotonic sodium chloride solution, interstitial salt solution [57] or lactated Ringer's solution should be given in maximum amounts in extensive burns.

Interest was renewed in electrolyte administration to burn patients by Rosenthal's [59] work, showing a lowering of the forty-eight hour mortality rate of scalded mice if isotonic sodium chloride solution was given orally within one hour following the burn. Sodium acetate, succinate, bicarbonate and lactate were as effective as sodium chloride. Intravenous administration was less effective. Potassium chloride increased the death rate, and when administered with sodium chloride antagonized the effects of the latter. Fox [60] then gave from 7 to 10 liters (10 to

---

57. Interstitial salt solution is made up of sodium chloride 6.5 Gm., sodium bicarbonate 2.5 Gm. and potassium chloride 0.18 Gm. per liter. It is not on the market at the present time, but lactated Ringer's solution can be substituted.

58. Harkins, H. N.: The Treatment of Burns, Springfield, Ill., Charles C Thomas, Publisher, 1942; Present Status of Problem of Thermal Burns, Physiol. Rev. **25**:531 (July) 1945. Walker, J., Jr.: The Pathologic Psysiology of the Extensive Superficial Burn, S. Clin. North America **26**:1488 (Dec.) 1946. Elkinton, J. R.; Wolff, W. A., and Lee, W. A.: Plasma Transfusion in Treatment of Fluid Shift in Severe Burns, Ann. Surg. **112**:150 (July) 1940.

59. Rosenthal, S. M.: Experimental Chemotherapy of Burns and Shock: Effects of Systematic Therapy on Early Mortality, Pub. Health Rep. **58**:512 (March 26) 1943.

60. Fox, L., Jr.: Oral Sodium Lactate in the Treatment of Burn Shock, J. A. M. A. **124**:207 (Jan. 22) 1944.

15 per cent of body weight) of chilled isotonic (one-sixth molar or 1.75 per cent) sodium lactate solution by mouth to a series of 23 burned patients with only 1 death, but considered a further evaluation of the treatment was needed. Moyer and his co-workers,[61] studying severely scalded dogs, found that massive transfusions of defibrinated blood and large amounts of sodium chloride and bicarbonate solution by stomach was the only form of therapy employed that prevented shock without inducing complications that were incompatible with life.

In a résumé of fluid and nutritional therapy of burns by a committee of the National Research Council [62] comments on electrolyte solutions for the prevention and treatment of burn shock, in the first forty-eight hours, were as follows: For burns of less than 10 per cent of the body surface, give 2,000 cc. of physiologic electrolyte solution [63] each twenty-four hours, preferably by mouth; if not, by vein. For burns of more than 10 per cent of the body surface, chief reliance should be placed on plasma or albumin or whole blood as indicated. Orally give 3,000 to 8,000 cc. of physiologic electrolyte solution [63] the first day, depending on the extent of the burn, and 3,000 cc. the second day.

These pertinent comments give present thought on the use of electrolyte solution in the early treatment of burn patients, and the literature should be watched for further developments along these lines. All observers caution against too much salt solution after the first forty-eight hours.

AMOUNT OF FLUID NEEDED TO CORRECT DEHYDRATION "ESTIMATES" OR "WORKING APPROXIMATES"

The amount of fluid needed for the correction of dehydration is not an entirely simple matter, since the degree of dehydration will vary in each case. But from the data on animal and human experiments and the response of carefully observed clinical cases an "estimate" or "working approximate" can be made of the initial amount of fluid to give. This is arrived at through a consideration of the history of the dehydration—how it developed, its duration, the possible volumes lost

61. Moyer, C. A.; Coller, F. A.; Iob, L. V.; Vaughan, H. H., and Marty, D.: A Study of the Interrelationship of Salt Solutions, Serum and Defibrinated Blood in the Treatment of Severely Scalded, Anesthetized Dogs, Ann. Surg. **120**:367 (Sept.) 1944.

62. Harkins, H. N.; Cope, O.; Evans, E. I.; Phillips, R. A., and Richards, D. W., Jr.: The Fluid and Nutritional Therapy of Burns, J. A. M. A. **128**:475 (June 16) 1945.

63. Physiologic electrolyte solution indicates a mixture of 2 parts of 0.9 per cent sodium chloride solution with 1 part of 1.3 per cent sodium bicarbonate solution. If sodium bicarbonate is not available 1.75 per cent sodium lactate solution may be substituted. These concentrations are approximately isotonic.

and whether any oral fluid was retained—and the thirst of the patient. From the physical examination note is made of the general appearance of the patient, the degree of inanition, evident loss of weight, dryness of the mouth and tongue, inelasticity of the skin and muscles, sunken eyes and the presence of edema. Laboratory studies tell about the volume and specific gravity of the first urine specimen, the erythrocyte and leukocyte count, the hemoglobin and hematocrit levels and the plasma chloride, protein, carbon dioxide combining power and the blood nonprotein nitrogen. This is a long list and more could be added, yet for the seriously dehydrated patient the good observer may use all of them for the initial estimation of fluid needs. The result of the initial fluid administration is shown by the general response of the patient and the changes in the urine and subsequent blood studies.

There is some risk in being too specific as to the amount of fluid to be given to a dehydrated patient, but it is well to know what working approximates have been developed from nutritional studies. The amount of water and electrolytes will vary with each case, and dependence must still be placed on the kidneys with a good supply of water to exercise some selection of the electrolyte ions needed and to excrete the excesses, thus maintaining satisfactory concentrations.

Years ago Rubner [64] stated that in starvation an animal can lose practically all of its glycogen and fat, half of its body protein, 40 per cent of its total body weight and still live, whereas the loss of 10 per cent of the water content results in serious disorder and the loss of from 20 to 22 per cent results in death. In 1923 Haden and Orr [65] recommended the administration of 1 Gm. of sodium chloride per kilogram of body weight as the initial dose of salt in the treatment of the toxemia of intestinal obstruction. While the salt restored body water and electrolytes rather than treated toxemia, theirs was the first attempt to place salt administration on a quantitative basis. In 1937 Falconer and Lyall [66] concluded that "in hypochloremia about 20 grammes (from 15 to 30 grammes) of salt are required on the average to raise the plasma chlorides by 100 mg. per 100 cc."

In 1938 Coller and his co-workers,[45b] from a quantitative study of administration of salt to surgical patients with hypochloremia from electrolyte fluid loss, suggested for clinical purposes that for each

64. Rubner, M.: Die Beziehungen der atmosphärischen Feuchtigkeit zur Wasserdampfabgabe, Arch. f. Hyg. **11**:137, 1890-1891.

65. Haden, R. L., and Orr, T. G.: Chemical Changes in the Blood of Man After Acute Intestinal Obstruction, Surg., Gynec. & Obst. **37**:465 (Oct.) 1923.

66. Falconer, M. A., and Lyall, A.: The Requirements of Sodium Chloride, Brit. M. J. **2**:1116 (Dec. 4) 1937; Blood Chemistry in Intestinal Obstruction, Lancet **2**:1472 (Dec. 25) 1937.

100 mg. per hundred cubic centimeters the plasma chlorides need to be raised to reach the normal (560 mg. per hundred cubic centimeters) the patient should be given 0.5 Gm. of sodium chloride per kilogram of body weight. In 1942 Power, Pedersen and Maddock [45a] reported their clinical experience with this formula on patients admitted to the hospital with depleted body electrolytes, mainly as a result of vomiting. Their conclusion was that the replacement formula was satisfactory in the patients of good or fair general condition who had suffered an acute or recent loss of gastrointestinal fluid. Patients who had a more chronic illness and whose general condition was poor frequently failed to utilize the sodium chloride in a normal fashion, most commonly depositing the salt and water in interstitial compartments and thus not increasing their plasma electrolytes to normal, but occasionally excreting the salt in the urine even though the plasma concentration was low. These patients of more chronic illness often did not have a history of recent or severe vomiting, and their lowered values of plasma chlorides seemed to have resulted from a long-standing or chronic sodium chloride deficiency or loss. Also, these patients commonly had one or more of the factors setting the background for the development of edema,[38] that is, general malnutrition, low plasma proteins, sepsis, severe hemorrhage, profuse serous drainage and severe renal or hepatic damage. Since considerable caution has to be used in giving more than moderate amounts of saline solution to such patients and because the plasma chloride level is not always a good index of electrolyte depletion, Coller and his co-workers [67] have advised that this formula not be used.

As suggested by Rubner's comment, usable working approximates on the amount of fluid needed to correct dehydration have been developed from body weight changes during dehydration. Studying human subjects Coller and Maddock [29] found that all of the common clinical observations of serious dehydration were present when persons had lost an amount of fluid equal to 6 per cent of their body weight. They suggested the use of this figure in estimating the amount of fluid needed to overcome dehydration deficits. From data on animals and patients, Darrow [20] estimated ". . . about one-third (8 per cent) of the extracellular electrolyte is lost in marked dehydration, and for restoration in such cases he estimates an amount of salt present in physiologic solution of sodium chloride equal to one-fifteenth (6.6 per cent) to one-twelfth (8.3) per cent of the body weight (about 70 cc. per kilogram of body weight) is required. Smaller amounts are needed in mild cases."

An example using the body weight data [29] of an estimation of the amount and kind of fluid to be given during the first twenty-four

67. Coller and others.[19] Moyer.[39]

hours to a 60 Kg. patient dehydrated because of gastrointestinal fluid loss would be as follows:

Water for vaporization	1,500 cc.	5 or 10% dextrose in water or protein hydrolysate solution
Water for urine	1,500 cc.	
Electrolyte fluid for dehydration,	3,600 cc.	5% dextrose in isotonic sodium chloride solution or Ringer's solution or lactated Ringer's solution as indicated
Total	6,600 cc.	

It should be emphasized that larger or smaller amounts of fluid might be needed, depending on the size of the patient, the degree of dehydration, the presence of fever requiring more water for vaporization, the presence of a previous retention of waste materials and/or a decreased concentrating power of the kidneys—both requiring more water for renal function—or a continued abnormal loss of fluid as by vomiting.

TABLE 7.—*Composition of Parenteral Fluids Supplying Water and Electrolytes and Comparison with Blood Plasma*

	Grams/Liter		Milliequivalents/Liter		
Isotonic sodium chloride solution	Sodium chloride	9.0	Sodium 155	Chloride 155	
Ringer's solution	Sodium chloride	8.6	Sodium 148	Chloride 148	158
	Potassium chloride	0.3	Potassium 4	Chloride 4	
	Calcium chloride	0.33	Calcium 6	Chloride 6	
Lactated Ringer's solution	Sodium lactate	2.5	Sodium 22	Lactate 22	111
	Sodium chloride	6.0	Sodium 103	Chloride 103	
	Potassium chloride	0.3	Potassium 4	Chloride 4	
	Calcium chloride	0.2	Calcium 4	Chloride 4	
Hypotonic sodium chloride solutions	Sodium chloride	4.5	Sodium 78	Chloride 78	
	Sodium chloride	6.0	Sodium 103	Chloride 103	
Blood plasma, mEq/L	Sodium	142	Chloride	103	
	Potassium	5	Bicarbonate	27	
	Calcium	5	Phosphates	2	
	Magnesium	3	Sulfates	1	
			Organic acid	6	
	Total	155	Protein	16	
			Total	155	

COMMON SOLUTIONS USED TO CORRECT DEHYDRATION

The electrolyte composition of the fluids commonly employed in the treatment of dehydration and their comparison with blood plasma is shown in table 7. These fluids and a few special solutions will be briefly discussed.

Isotonic Sodium Chloride Solution.—This solution has had the most widespread use for replacing deficits of extracellular fluid and treating both alkalosis and acidosis. Now more thought is given to the exact ions needed and the kidneys are thus saved from having to make all the selection and do extra work when function may be poor because of the disease conditions. Also, the shift of fluid between the intracellular and extracellular compartments is considered to be more in the right direction when fluids balanced to meet the needs are given.

In milliequivalents isotonic solution of sodium chloride has the same amount of sodium as chloride, but compared to the concentration of these substances in blood plasma, see table 7, it contains relatively more chloride. It should be used for cases in which the chloride loss is greater than the sodium loss, such as the vomiting of pyloric obstruction, or when the chloride and sodium losses are more nearly equal, as in vomiting from duodenal, jejunal or ileal obstruction or the replacement of gastrointestinal fluid aspirated by Wangensteen or Miller-Abbott tube suction.

Ringer's Solution.—This solution has a small amount of potassium and calcium chloride in addition to a good quantity of sodium chloride, but it is difficult to prove from clinical use that it has any advantage over isotonic sodium chloride solution. It may have a little less tendency to produce edema than the latter.[45c] However, because in dehydration there is usually some loss of calcium and potassium besides sodium and chloride ions I prefer it to the isotonic sodium chloride solution. The indications for its use are the same as given for isotonic sodium chloride solution. Ringer's solution does not contain sufficient potassium to replace a serious deficiency of that substance.

Lactated Ringer's Solution.—The 2.5 Gm. of sodium lactate in this solution provides an excess of sodium after the lactate is metabolized.[64] It has been pointed out that with serious impairment of hepatic function the splitting off of the lactate radical, which is part of the metabolic process, may be interfered with,[68] so that in such cases sodium bicarbonate is a better substance where an excess of sodium is needed. In general, lactated Ringer's solution is desirable in practically all cases where acidosis is present or possible, and its use was discussed in the paragraphs under "specific examples of water and electrolyte loss."

Hypotonic Sodium Chloride Solutions (Sodium Chloride 4.5 to 6.0 Gm. per Liter).—Coller and his co-workers [37] recently advocated the greater use of these solutions because of the relatively high incidence of "salt intolerance following a general anesthesia." In order to provide more freely available water, and not to draw water from the intracellular compartment, they [19] stated that "if intravenous infusion is indicated in the post-operative care of the surgical patient, hypotonic solutions, 0.45 per cent NaCl, or better 0.38 per cent NaCl plus 0.11 per cent $NaHCO_3$, should replace the 'isotonic solutions' commonly in use."

Isotonicity was devised primarily to prevent destruction of red blood cells as the fluid was administered. Nine grams of sodium chloride per liter does this, but since almost always 5 per cent dextrose is also added to the solution, less than 9 Gm. of sodium chloride is needed

68. Minot, A. S.; Dodd, K., and Saunders, J. M.: Acidosis of Guanidine Intoxication, J. Clin. Investigation **13**:917 (Nov.) 1934.

for isotonicity. Electrolyte solutions used should fit not the cells of the blood but the concentration of the electrolytes in the blood and interstitial spaces. Therefore sodium chloride solutions of about 5 to 6 Gm. per liter are more nearly correct for this purpose than the present 9 Gm. solutions.

SPECIAL SOLUTIONS FOR SEVERE ALKALOSIS AND ACIDOSIS

It has long been suggested that in the extreme alterations of acid-base balance the initial treatment should be a correction of the severe alkalosis or acidosis to a moderate or mild alkalosis or acidosis, and the substances discussed below have been used for that purpose. Immediately after the extreme of the compositional change has been corrected, then isotonic sodium chloride solution, Ringer's solution or lactated Ringer's solution should be given as indicated to correct the remaining extracellular water and electrolyte deficiency. In this way the most needed ions are immediately provided, the kidneys which may well have a disturbance in their selective function have a lesser load and severe alterations in extracellular fluid patterns are more quickly corrected.

Hydrochloric Acid (for Severe Alkalosis).—Cullen [69] proposed the use of hydrochloric acid for an alkalosis of over 90 volumes per hundred cubic centimeters of plasma carbon dioxide and makes up the solution by the following rule: "The amount of concentrated HCl (36 Per cent) = Vol. % CO_2 excess (over 70) x 0.028 x wt. (kilo). Add the acid to 0.9% NaCl solution so that there is never more than 5 cc. of concentrated acid per 100 cc. of saline." Moyer [39] presented this calculation as follows: Take 0.063 cc. of concentrated hydrochloric acid (36 per cent) per kilogram of body weight for each milliequivalent the base bicarbonate is above 30 milliequivalents per liter of plasma; dilute with 0.9 per cent sodium chloride solution so that not more than 5 cc. of concentrated acid is present in 100 cc. of sodium chloride solution. This solution is then given slowly and the injection stopped if dyspnea appears. If the necessary laboratory data are not available and severe alkalosis is present, Moyer [39] advised giving a one-hundredth molar solution of hydrochloric acid until the carpopedal spasm and the hypopnea are relieved.

Ammonium Chloride (for Severe Alkalosis).—A 0.9 per cent solution has been used, and in an emergency when the usual laboratory data are not available Moyer [39] stated that ammonium chloride may be given until the carpopedal spasm and the hypopnea are relieved. Zintel,

69. Cullen, G. E.: The Factors Governing Fluid Therapy in the Treatment of Enteritis, Ohio State M. J. **32**:509 (June) 1936.

Rhoads and Ravdin [70] have treated surgical patients with severe alkalosis with 2 per cent ammonium chloride in 0.9 per cent sodium chloride solution intravenously and have noted that it was effective in lowering the serum carbon dioxide at the rate of approximately 1 volume per cent for each gram administered to adults of medium size and weight.

Sodium Bicarbonate (for Severe Acidosis).[71]—Hyperpnea usually indicates when sodium bicarbonate, or sodium lactate, is advisable. A 1.3 per cent solution is isotonic, and for administering sodium bicarbonate in severe acidosis Moyer [39] used 60 mg. of sodium bicarbonate per kilogram of body weight of the 1.3 per cent solution for each milliequivalent the base bicarbonate is below 25 milliequivalents per liter of plasma.

Another method of sodium bicarbonate calculation is presented by the equation of Hartmann and Senn [72]:

$$mM = \frac{(60\text{-}CO_2)\ 0.7\ W}{2.24}$$

In this equation, 1 mM is millimols of sodium bicarbonate or sodium lactate. One millimol of sodium bicarbonate is 0.084 Gm. One millimol of sodium lactate is contained in 1 cc. of molar sodium lactate; CO_2 is the serum carbon dioxide content in volumes per cent, and W is the body weight in kilograms.

If the plasma carbon dioxide is not known, Darrow [20] stated that 5 millimols of sodium bicarbonate (0.4 Gm.) per kilogram of body weight is a safe dose in severe acidosis, and Moyer said that the 1.3 per cent solution may be given intravenously until the hyperpnea and dyspnea are relieved, which usually does not take more than 2 liters for an adult.

Sterile ampules of sodium bicarbonate are now available and may be added to sterile water to make up a 1.3 per cent solution for intravenous administration. For the same use the sterile sodium bicarbonate may be added to the commonly used flasks of 5 per cent dextrose in water to make a 2 to 5 per cent sodium bicarbonate concentration.[20]

Sodium Lactate (for Severe Acidosis).—A 1.75 per cent solution is isotonic. Molar sodium lactate solution is available in ampules, but

70. Zintel, H. A.; Rhoads, J. E., and Ravdin, I. S.: The Use of Intravenous Ammonium Chloride in the Treatment of Alkalosis, Surgery **14**:728 (Nov.) 1943.

71. Darrow, D. C.: Medical Progress: Body-Fluid Physiology; Relation of Tissue Composition to Problems of Water and Electrolyte Balance, New England J. Med. **233**:91 (July 26) 1945.

72. Hartmann, A. F., and Senn, M. J. E.: Studies in the Metabolism of Sodium *r*-Lactate: II. Response of the Human Subjects with Acidosis to the Intravenous Injection of Sodium *r*-Lactate, J. Clin. Investigation **11**:337 (March) 1932.

is diluted to one-sixth molar concentration with distilled water or 0.9 per cent sodium chloride solution for parenteral use.

Hartmann [72] recommended a calculation of the dosage by the formula presented in the paragraphs on sodium bicarbonate, and, using the one-sixth molar sodium lactate solution, recommended that one half should be given intravenously and one half subcutaneously.

As mentioned previously, sodium lactate should not be used in cases of severe hepatic damage,[68] since it may then not be split and the sodium not become available for the treatment of the acidosis. Sodium bicarbonate is better in such instances.

RENAL INSUFFICIENCY DURING AND FOLLOWING SEVERE DEHYDRATION

Insufficient renal function during and immediately following severe dehydration has been a problem, and in general the profound changes that go with oliguria or anuria are not realized. The disturbance in most instances is due to a number of factors.

In severe dehydration the reduction in blood plasma volume is accompanied by a lowered blood pressure and consequently a lowered filtration pressure through the kidneys. At the same time an increase in the osmotic pressure and in the viscosity of the blood plays an important additional part in greatly reducing the volume flow of blood through the kidneys as well as other organs.[73] Utheim [74] was able to measure in dehydrated infants a tenfold reduction in the volume flow of blood.

A further renal factor is that of actual disease in the kidneys altering renal controls. This disease may have been previously present as chronic nephritis, or it may have come on as a disturbance of function secondary to severe degrees of dehydration. Gamble [22] well presented the possible alterations in renal controls as (1) "retentions" due to inability to excrete substances like phosphates and sulfates and possibly organic acid radicals, and (2) the opposite errors of control in the way of "inability to reabsorb." In regard to the latter Gamble pointed out that plasma values are not established by the kidneys by a direct removal of surplus but by reabsorption of what is needed from tubular fluid. With permanent or temporary impairment of renal function sodium in particular and chloride ions to a lesser extent may thus be lost through the kidneys even though they are needed to keep up normal concentration in the plasma.

---

73. Gamble.[12] Hartmann, A. F.: Chemical Changes Occurring in the Body as a Result of Certain Diseases, Am. J. Dis. Child. **35**:557, (April) 1928. Abbott, W. E.: Fluid, Electrolyte and Nutritional Problems in Surgery, S. Clin. North America **26**:1330 (Dec.) 1946.

74. Utheim, K.: Advanced Chronic Nutritional Disturbances in Infancy, J. Metabolic Research **1**:803 (June) 1922.

A third factor in renal insufficiency in relation to dehydration is lack of water, and this properly has been emphasized for years. The kidneys use water to excrete body waste materials in solution. With insufficient water intake by mouth the value of the parenteral administration of dextrose solutions in water cannot be too highly stressed. They provide easily available water to be spent as needed, the carbohydrate reduces protein loss, and the maximum of this effect is gained by supplying 100 Gm. daily for an adult. Further, the metabolism of this dextrose prevents ketone acid formation and relieves the kidneys of having to excrete these abnormal products at a time of many other stresses. A great deal is thus gained by providing a good intake of water and dextrose.[22]

DAILY REPLACEMENT OF GASTROINTESTINAL FLUID LOSS

In the previous paragraphs dehydration as a result of abnormal gastrointestinal fluid loss has been discussed and treatment recommended. But for patients in hospitals, modern medical practice requires that dehydration not be allowed to develop. If gastrointestinal fluid losses are occurring, daily replacement of the water and electrolytes is essential. By means of collecting bottle apparatus and proper house staff and nursing skill the amount of the daily drainage is known and recorded. Common sources of such drainage are from aspiration of gastrointestinal fluid through Wangensteen or Miller-Abbott tubes, or one of their modifications, or from biliary tract drainage. Less common sources are pancreatic and intestinal fistula drainage and from ileostomies.

The concentration of the important electrolytes in these secretions and in some of the replacement solutions is shown in table 6. As mentioned previously there is some variation in the electrolyte pattern of any given gastrointestinal juice, particularly in vomitus, and there largely dependent on the amount of fluids taken shortly before the vomiting. The greater acid nature of fundus gastric juice over that of pylorus gastric juice is shown. Vomitus carries away much more chlorides than sodium. From there on down the sodium loss is greater than the chloride loss, the difference being most pronounced in ileal and colon secretion.

Table 6 shows importantly that the concentration of the electrolytes in isotonic sodium chloride solution or Ringer's solution is nearly always greater than their concentration in the digestive fluids. It was therefore suggested in 1937 [75] that volume for volume replacement of a

75. (*a*) Dick, V. S.; Maddock, W. G., and Coller, F. A.: Sodium Chloride Content of Gastro-Intestinal Secretions, Proc. Soc. Exper. Biol. & Med. **37**:318 (Nov.) 1937. (*b*) Nadler, S. B.: The Use of Parenteral Fluids, Surgery **1**:964 (June) 1937.

digestive fluid loss with isotonic sodium chloride solution or Ringer's solution would maintain sodium and chloride electrolyte concentrations at nearly normal levels. Subsequent studies by Coller and others [76] have found this approximation to work satisfactorily. When continuous gastrointestinal suction is started it is well to give a liter of isotonic sodium chloride solution or Ringer's solution on the first day of the suction in order to forestall an appreciable initial drop in electrolytes. If it is apparent that the immediate suction drainage loss is very large, then more than a liter of these solutions should be given the first day. From then on, volume for volume replacement works well.

For biliary tract drainage, which carries away more sodium than chloride ions, lactated Ringer's solution can be used for replacement on the volume for volume basis.

Hoffman [77] recently recommended the use of the volume for volume replacement method, and Moyer [89] employed it but gave an 0.45 to 0.6 per cent sodium chloride solution instead of the isotonic sodium chloride solution for gastric drainage with free hydrochloric acid in it.

A somewhat different way of calculating daily replacement of lost electrolytes comes from the pointed out fact that the sodium chloride concentration of the gastrointestinal tract fluids, calculated from the chloride concentration, is about 5 Gm. per liter.[75a] Wangensteen [82] employed this principle by giving 5 Gm. of salt in the form of isotonic sodium chloride solution for each liter of fluid aspirated by continuous gastrointestinal suction.

These approximates for the daily replacement of gastrointestinal fluid loss work rather well in the average case, but special care is necessary when the amount of fluid abnormally drained from the gastrointestinal tract is large, which means more than 2,000 cc. daily. Then, along with the volume for volume replacement rule, frequent chemical studies of the blood should be made as checks and alkalosis or acidosis looked for. Although special solutions are not often needed, they are of value in instances of severe alkalosis or acidosis.

POTASSIUM

In 1946 Govan and Darrow [42] reported a reduction in the mortality rate of infants with diarrhea from 17 in 53 to 3 out of 50 by treating their dehydration with a solution containing potassium chloride in addition to sodium chloride, sodium lactate and dextrose. This striking result is a further example of the long line of outstanding pioneer work done by pediatricians on water and electrolyte problems, and the

76. Bartlett, Bingham and Pedersen.[44a] Coller and others.[45b]

77. Hoffman, W. S.: The Estimation of Water and Salt Requirements in the Surgical Patient, S. Clin. North America **27**:217 (Feb.) 1947.

surgeons and others interested in such problems owe much to their basic studies. The surgeons are more concerned with the abnormal loss of fluid from the upper end of the gastrointestinal tract, but are confronted with severe dehydration probably also involving important losses of potassium.

In an article on the retention of electrolyte during recovery from severe dehydration due to diarrhea Darrow [78] reviewed important concepts on potassium as follows: The demonstration of the loss of potassium as well as sodium and chloride in severe diarrhea is not new. An extensive disintegration of cells has long been known to occur, and the loss of potassium was relatively greater than the loss of nitrogen. Holt and his co-workers [79] in discussing replacement of this loss said that not only water and sodium are needed, but also potassium and magnesium, and that a better solution could be devised than isotonic sodium chloride solution or Ringer's solution. But over many years adequate amounts of potassium were not given, probably because of two reasons: (1) the realization that a rise in the concentration of plasma potassium could produce death by stopping the heart, and (2) cellular membranes were considered to be relatively impermeable to cations, and hence loss of potassium was considered to be caused by some fundamental change within the cells. The latter proposition is no longer tenable, since in the past ten years it has been shown that intracellular fluid contains some sodium, and potassium readily crosses the cellular membranes. Intravenous potassium can produce heart block when serum concentrations reach a level of 10 to 12 millimols per liter,[80] but if the dosage is given slowly and the total amount is not too high this complication can be avoided. The kidneys can excrete potassium in concentrations of over 100 millimols per liter, so that a good output of urine is an added safeguard against a dangerously high potassium concentration.

The concentration of electrolytes used by Darrow [78] in the treatment of severe dehydration produced by diarrhea in infants is potassium chloride 2 Gm., sodium chloride 3 Gm., one-sixth molar sodium lactate solution 250 cc. and water 500 cc. About 80 cc. of such a solution per kilogram of body weight are given to the infants over a period of four to eight hours. Considering the possible complications of potassium

78. Darrow, D. C.: The Retention of Electrolyte During Recovery from Severe Dehydration Due to Diarrhea, J. Pediat. **28**:515 (May) 1946.

79. Holt, L. E.; Courtney, A. M., and Fales, H. L.: The Chemical Composition of Diarrheal as Compared with Normal Stools in Infants, Am. J. Dis. Child. **9**:213 (March) 1915.

80. Winkler, A. W.; Hoff, H. E., and Smith, P. K.: Electrocardiographic Changes and Concentration of Potassium in Serum Following Intravenous Injection of Potassium Chloride, Am. J. Physiol. **124**:478 (Nov.) 1938.

therapy, heart block and erythema with desquamation, Darrow's [42] article should be reviewed before potassium is given. Concerning the use of potassium in surgical cases Darrow stated, ". . . the treatment of acidosis should not be undertaken without considering the effect on the cells; and probably the use of the salts of both sodium and potassium is indicated in other types as well as in diarrheal acidosis." [81]

FAT EMULSIONS FOR PARENTERAL NUTRITION

The chief advantage in the use of fat in parenteral nutrition lies in its high caloric value, and to obtain this effect experimental studies have been carried out since the beginning of this century.[82] The problem of breaking fat down to minute droplets for intravenous administration has been solved by the use of emulsifiers employing pressures up to 5,000 pounds per square inch. The result is fat particles varying in size from a fraction of a micron to 2 microns in diameter, which is exceedingly small compared with red blood cells of 6 to 9 microns and leukocytes of 5 to 15 microns. The stabilization of a fat solution, which is the prevention of its breakdown into larger particles on standing or handling, has been difficult, and many substances have been tried. Surface-active or wetting agent types of emulsifiers are thought to be effective by imparting an electric charge at the periphery of the droplet, while the hydrophilic colloid types are believed to envelop the droplet with a protective film of the colloid material.

The use of seemingly satisfactory fat emulsions is still in an experimental phase. Some animals studied have died with mechanical block interference of function in the liver, lungs and kidneys, but this result is not common. As to the utilization of the injected fat, the different criteria have shown variable success. Dunham and Brunschwig,[82] using emulsions of olive oil and lard oil, found insufficient change in the iodine and saponification numbers of depot fat in the omentum of dogs to indicate appreciable physiologic storage of the infused fat, and a protein-sparing effect was not demonstrated. In some of their animals, a third criteria, that of changes in the melting point of depot fat, did suggest some storage of infused fat. The infusions of fat produced severe secondary anemia in the dogs, from which they recovered after the infusions were discontinued.

McKibbin and his associates [83] stated the belief that coconut oil was superior to corn oil as an infusion fat in dogs, and, from an improve-

81. Harkins and others.[62] Darrow.[78]

82. Dunham, L. J., and Brunschwig, A.: Intravenous Administration of Fat for Nutritional Purposes, Arch. Surg. **48**:395 (May) 1944.

83. McKibbin, J. M.; Pope, A.; Thayer, S.; Ferry, R. M., Jr., and Stare, F. J.: Parenteral Nutrition: Studies on Fat Emulsions for Intravenous Alimentation, J. Lab. & Clin. Med. **30**:488 (June) 1945.

ment in weight and nitrogen retention, and no evidence of the storage of coconut oil by changes in saponification and iodine numbers of depot fat, stated the belief that the fat is utilized by the dog for energy.[84]

Emulsions of fat have been given intravenously to human beings—infants, children and adults—with reported beneficial results,[85] but much greater animal experimental and clinical experience is necessary before common use can be considered.

VITAMINS

The need for vitamins by surgical patients is mainly a part of their general need for excellent nutrition to combat a disease or injury. Patients who have been eating a good diet usually present no problem and come to operation in good shape. On the other hand, many ill patients require attention to vitamin needs just as they do for their protein and other food deficiencies.

The factors predisposing to the development of vitamin deficiency diseases as listed by Wilbur [86] can be briefly presented as follows:

1. Inadequate intake of vitamins as a result of
   (*a*) Economic difficulties
   (*b*) Ignorance, eccentricities, poor dietary habits, fadism and chronic alcoholism
   (*c*) Anorexia, dysphagia, dyspepsia and pain
   (*d*) Long-continued use of a therapeutic diet which is nutritionally inadequate
2. Increased need for vitamins which occur during periods of
   (*a*) Rapid growth, particularly infancy and childhood

---

84. McKibbin, J. M.; Ferry, R. M., Jr., and Stare, F. J.: Parenteral Nutrition: II. The Utilization of Emulsified Fat Given Intravenously, J. Clin. Investigation **25**:679 (Sept.) 1946.

85. Holt, E., Jr.; Tidwell, H. C., and Scott, T. F. M.: The Intravenous Administration of Fat: Practical Therapeutic Procedure, J. Pediat. **6**:151 (Feb.) 1935. Narat, J. K.: Ueber Intravenöse Fetteinspritzungen, Arch. f. klin. Chir. **187**:795, 1937; Observations on Parenteral Administration of Fat Emulsion, Am. J. Digest. Dis. & Nutrition **4**:107 (April) 1937; Parenteral Use of Fat Emulsions in Urosepsis, Urol. & Cutan. Rev. **42**:17 (Jan.) 1938; New Substitute for Ketogenic Diet, J. Urol. **39**:75 (Jan.) 1938. Krainick, H. G.: Fortschritte der parenteralen Lipoidtherapie, Therap. d. Gegenw. **81**:60 (Feb.) 1940. Valledor, T.: Lipothérapie intraveineuse dans le traitement des dystrophies graves du nourrisson, spécialement dans les dystrophies d'origine tuberculeuse, Arch. de med. d. enf. **36**:276 (May) 1933. Gordon, H. H., and Levine, S. Z.: Respiratory Metabolism in Infancy and in Childhood: Effect of Intravenous Infusions of Fat on Energy Exchange of Infants, Am. J. Dis. Child. **50**:894 (Oct.) 1935. Clarke, D. E., and Brunschwig, A.: Intravenous Nourishment with Protein, Carbohydrate and Fat in Man, Proc. Soc. Exper. Biol. & Med. **49**:329 (March) 1942.

86. Wilbur, D. L.: The Role of the Gastro-Intestinal Tract in Conditioning Deficiency Diseases, M. Clin. North America **27**:519 (March) 1943.

(*b*) Pregnancy and lactation
(*c*) Increased metabolic activity as in hyperthyroidism
(*d*) Acute and chronic infections

3. Abnormalities in function of gastrointestinal tract
   (*a*) Inadequate intake of food and vitamins
   (*b*) Loss of essential digestive secretions or of food as by vomiting, fistulas or diarrhea
   (*c*) Lack or diminished production of essential endogenous substances
      (1) Stomach
         (*a*) Loss of intrinsic factor of Castle—pernicious anemia
         (*b*) Some gastric resections—macrocytic anemia
         (*c*) Loss of hydrochloric acid—hypochromatic anemia, interference with absorption of vitamin B and C (?)
      (2) Liver: Failure of bile to reach intestines—poor fat and vitamin A absorption, interference with vitamin K metabolism
      (3) Pancreas: Extensive atrophy produces clinical picture like sprue
   (*d*) Inadequate intestinal absorption as a result of obstructions, atrophy or disease of mucosa, or various short circuitings: pellagra, impairments characteristic of sprue, anemia simulating pernicious anemia
4. Alterations in metabolism of vitamins
   (*a*) Extensive hepatic damage
      (1) Deficiency of vitamin A
         (*a*) Liver essential in conversion of carotene to vitamin A
         (*b*) Liver important storehouse of vitamin A
      (2) Faulty metabolism of vitamin K
         (*a*) Liver indispensably involved in metabolism of vitamin K and prothrombin formation
         (*b*) Liver may fail to use vitamin K supplied
   (*b*) Gastrointestinal tract disease
      (1) May destroy vitamins by bacterial or chemical action
      (2) Decrease in synthesis of certain vitamins, possibly vitamin K and certain components of B complex (?)

TABLE 8.—*Vitamins*

	Vitamin	Daily Maintenance Dose, I. U.	Amounts Suggested with Deficiency, I. U.
A		5,000	25,000-100,000
D		500-1,000	2,000-5,000
		Mg.	Mg.
B	Thiamine chloride	1.2-2.3	10-50
	Riboflavin	1.8-3.3	5-10
	Niacinamide	15-25	250-500
	Calcium pantothenate	5-10	50-100
	Pyrodoxine hydrochloride	1-5	5-100
C		75	100-1,000
K		0.1	5-10

From the point of view of the parenteral administration of vitamins to surgical patients, usually B complex and C are given frequently, and K in cases of present or possible hepatic damage. Table 8, calculated

from several sources,[87] shows the daily maintenance dose and the amounts suggested to prevent or treat deficiencies. In the column for deficiencies the higher figures are for the severest disease. It is interesting to note the great excess of the treatment quantities over the maintenance dose. This has come about largely because of uncertainty as to just how much is needed and, secondly, because no harm seems to develop from the large amounts given over relatively short periods of time.

Vitamin A is found deficient in some jaundice patients and should be given, along with the other vitamins, to these and other ill patients. Preparations for parenteral administration are becoming available.

For the administration of vitamin B complex and vitamin C the various pharmaceutic houses have material for intramuscular and intravenous administration reasonably covering the quantities shown in table 8. A product should be selected and added to the daily dextrose and/or electrolyte solutions given intravenously. It should not be mixed with protein hydrolysate fluids, since incompatabilities may develop. While all components of vitamin B are needed, thiamine chloride is considered by some to be particularly important because it is concerned with carbohydrate metabolism and rapidly becomes depleted in pathologic conditions. Wilder[87f] suggested not less than 1 mg. for each 50 Gm. of sugar, while a common amount is at least 10 mg. daily when dextrose solutions are given. Gordon[87m] stated that under conditions of the most pronounced thiamine deficiency the dose does not need to exceed 25 to 30 mg. daily. For vitamin C, 500 mg. per day is a good figure in the preoperative and postoperative hospital period.

87. (*a*) Clausen, S. W.: Vitamin A Deficiency, M. Clin. North America **27**: 349 (March) 1943. (*b*) Warkany, J.: Vitamin D Deficiency (Rickets), ibid. **27**:361 (March) 1943. (*c*) Warner, E. D.: Vitamin K Deficiency, ibid. **27**:371 (March) 1943. (*d*) Smith, D. T.: Nicotinic Acid Deficiency, ibid. **27**:379 (March) 1943. (*e*) Butler, R. E.: Riboflavin Deficiency, ibid. **27**:399 (March) 1943. (*f*) Wilder, R. M.: Thiamine Deficiency, ibid. **27**:409 (March) 1943. (*g*) Butler, A. M.: Vitamin C Deficiency, ibid. **27**:441 (March) 1943. (*h*) Bean, W. B.: Adenylic Acid in Nutrition, ibid. **27**:483 (March) 1943. (*i*) Lund, C. C., and Crandon, J. H.: Nutrition As It Affects Wound Healing, ibid. **27**:561 (March) 1943. (*j*) Wolfer, J. A., and Hoebel, F. C.: The Significance of Cevitamic Acid Deficiency in Surgical Patients, Surg., Gynec. & Obst. **69**:745 (Dec.) 1939. (*k*) Crandon, J. H.; Lund, C. C., and Dill, O. B.: Vitamin C Deficiency in Otherwise Normal Adults: Experimental Human Scurvy, New England J. Med. **222**:748 (May 2) 1940; **223**:353 (Sept. 5) 1940. (*l*) Wolfer, J. A.; Farmer, C. J.; Carroll, W. W., and Manshardt, D. O.: An Experimental Study in Wound Healing in Vitamin C Depleted Human Subjects, Surg., Gynec. & Obst. **84**:1 (Jan.) 1947. (*m*) Gordon, E. S.: Nutritional and Vitamin Therapy in General Practice, ed. 3, Chicago, The Year Book Publishers, Inc., 1947.

As soon as food and fluid can be taken by mouth a moderate amount of all the vitamins should be given orally while the patient is in the hospital, during a four to eight week recovery period, and longer for those presenting continued nutritional problems.

COMMENT

The ability to administer nutritional substances to patients by parenteral routes is one of the great advances in modern medicine. By no means have all problems in this treatment been solved, but considerable basic information is available. Its application has enabled the all-pervading body fluid, the real living environment of the cells of thousands of patients to be kept more nearly normal under conditions of disease. Being of particular value in the support of the seriously ill patient, the selection of amounts and kinds of material approximating the needs of these patients has saved countless lives.

LESIONS OF PATELLAR CARTILAGE AS A CAUSE OF INTERNAL DERANGEMENTS OF THE KNEE

CHARLES W. PEABODY, M.D.
AND
FRANK P. WALSH, M.D.
DETROIT

A CONSIDERABLE proportion of surgeons will at one time or another be meeting either for treatment or diagnosis of the disorder which is termed internal derangement of the knee joint. For many years we have obtained the impression that out of teaching and general medical knowledge there was a too persistent and frequent obscurity as to the various pathologic conditions, in their recognition and treatment, that make up this type of disorder. Also, in the approach and planning of the operative treatment that should so successfully resolve the complaint or disability, the status was perhaps analogous to another one of several decades ago when the operative listing in the majority of abdominal operative cases was "exploratory laparotomy" because of handicaps in preoperative recognition of pathologic processes and the appropriate surgical planning for each.

In recent years, particularly in consequence of extensive professional contacts and associations in military service, it has appeared to us that the existence of one particular form of internal derangement of the knee joint has been unappreciated, either entirely or in its frequency of incidence, by a surprisingly large number of doctors. This entity is the deranging lesion of the cartilage of the patella, and this is the subject which we shall discuss.

REVIEW

The following outline lists the group of conditions which represent what might be called the intrinsic forms; in other words, those conditions which are the result of neither inflammatory nor neoplastic processes, nor of the residuals of fractures of the major bones. In this list, arranged roughly in order of frequency, the placement toward the top of patellar cartilage lesions may be noted.

1. Meniscus lesions, post-traumatic
2. Chondropathy patella
3. Osteochondritis dissecans

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

4. Joint mouse (simple loose body)
5. Recurrent dislocation patella
6. Ligamentous instability
7. Degenerative meniscus lesions
8. Ununited fracture tibial eminence
9. Hypertrophied synovial villi
10. Osteochondromatosis

TERMINOLOGY AND INCIDENCE

In the matter of terminology, we should like to labor a certain point; namely, that the term osteochondritis applied to this latter entity is a misnomer and undesirably misleading. The pathologic lesion is not at all one of bone but essentially of cartilage, and only at a very chronic and advanced stage is bony tissue involved eventually, and solely through exposure. Of most extreme rarity is the occurrence, analogous to that in the femoral condyle, of primary aseptic bone necrosis and subsequent chondral degeneration, to which the term osteochondritis applies. The occasional—and usually not functionally disturbing—incidence of congenital bipartite patella could be interpreted as osteochondritis by those unfamiliar with it. The following outline shows the evidence of three types.

1. Congenital: Chondromalacia; incidence, second and third decades
2. Traumatic: Chondritis, noninflammatory; incidence, third and fourth decades
3. Degeneration: Presenescent chondrosis; incidence, fourth, fifth or sixth decade

The relative incidence of lesions of the cartilage of the patella is a matter of variability as to the sources and conditions under which the clinical material may be drawn. An orthopedic surgical confrere, when functioning as a regional consultant in the Army, found this patellar cartilage disorder in a little over 18 per cent of fifty consecutive arthrotomies. In a somewhat larger series in the naval service, in which I myself either performed or directed the arthrotomy, the incidence was about 20 per cent. A high majority of the patients, with numerous medical examinations, had arrived on my service with the lesion undiagnosed, despite an adequate basis for it, and many had had unsuccessful operations with erroneous diagnosis.

This then might be looked on as the incidence rate for young men exposed to occupational or recreational hazards to the knee joint. On the other hand, in a general civilian orthopedic practice covering all age periods and activities I have found this lesion to have a higher incidence of recognition than any other internal derangement of function, and it is approaching first place in indications for arthrotomy.

PATHOLOGIC ASPECTS

1. Chondromalacia: embryologically imperfect cell differentiation with malacic degeneration, softening and disintegration, involving entire structure
2. Post-traumatic chondritis: an unhealed (because basically irreparable) crush or fracture of cartilage with spreading fibrillary degeneration
3. Presenescent chondrosis: more premature in cartilage of patella than any other joint, independent of inflammatory joint disease

From the accumulation of observations over the last decade, we have become entirely satisfied that these lesions of the patellar cartilage are subdivisible as to pathologic character into three separate forms.

To the first—and least frequent—the term chondromalacia, I believe, most properly applies. It is of congenital origin, with symptoms

Fig. 1.—Congenital chondromalacia. The specimen was obtained by complete surface resection of patella cartilage in a 14 year old girl. The natural hue was bluish white. Throughout the substance was soft and plushy, with areas of wrinkling and ridging, and the focal point of penetrating loss of substance. The patient was symptom free six years later.

manifest sometimes as early as the second decade, and usually mistaken for rheumatic disease. The process is not a localized one, but involves the entire structure of the cartilage of the patella. Grossly the entire surface will be lacking in normal hardness, but instead will be sort of "plushy" indenting or even wrinkling on pressure. Instead of a pearly white opalescence, it has a bluish tinge. The surface is uneven or undulating, and to varying extent there will be irregular areas of ragged fragmentation and loss of substance, some constituting the entire thickness down to underlying bone. Microscopically, the cell structure everywhere is that of imperfect differentiation to mature hyaline cartilage and consequent attritional degeneration and fibrillation.

The second type, strikingly different, constitutes the residual conditions and consequences of localized traumatic fracture or laceration at a point on the articular surface; elsewhere the surface is normal in appearance both in color and consistency. With hyaline cartilage having no power whatever of healing or repair, not only does the lacer-

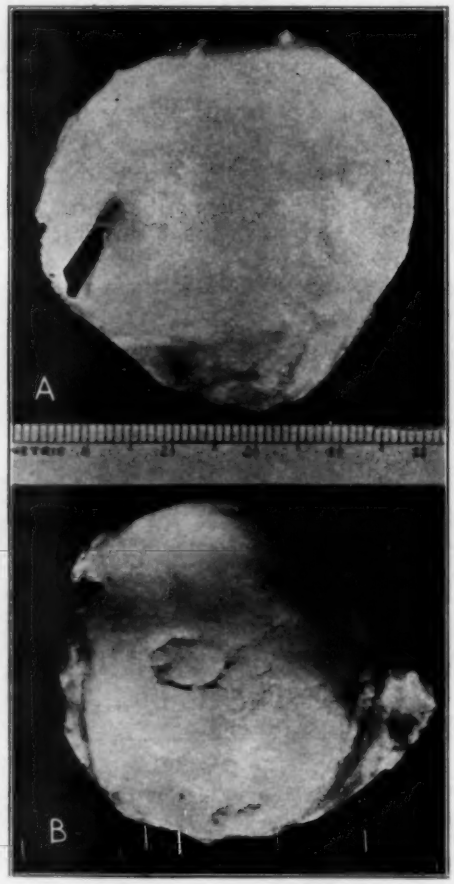

Fig. 2.—Traumatic chondritis. *A*, acute laceration (deep fissure) as indicated by the inserted stylet. The patella was removed on account of attendant degenerative chondrosis, as evident in multiple striae, scoring and surface erosions.

B, the specimen is a patella excised after an acute flap type laceration occurred as a final episode of repeated dislocations of the patella. Traumatic fissures adjacent, early origin and diffuse surface fibrillary degeneration may be noted.

ation persist but, under the attritional trauma of friction and compression, the defect gradually enlarges through fibrillary degeneration into widening fissures, fringes and even flaps, sometimes, though not usually, reaching the depth of the underlying bone.

The third type represents localized premature senescence of hyaline cartilage cell structure, to which that of the patella is peculiarly suscep-

tible, and might properly be termed degenerative patellar chondrosis. It usually is an isolated manifestation in the particular joint for a long period after its inception, and, though often to some degree bilateral, it will be the only manifestation for many years of presenescent

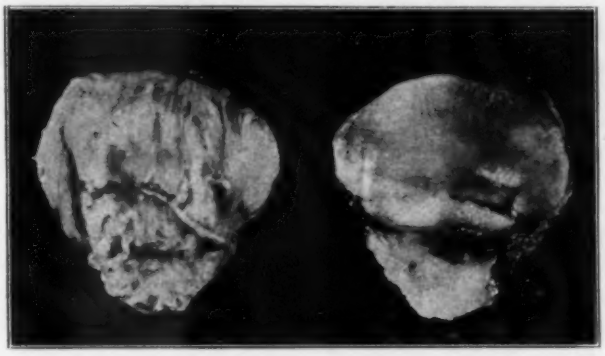

Fig. 3.—An excised patella, with the deep surface on the left and the subcutaneous surface on the right. This was removed from a woman in her forties with long-standing crepitation of the knee and acute comminuted fracture without separation. Ancient striae and fissures throughout the cartilaginous surface, in addition to fracture lines in the inferior half, may be noted. Conservative treatment would have doomed the patient to increasing disability.

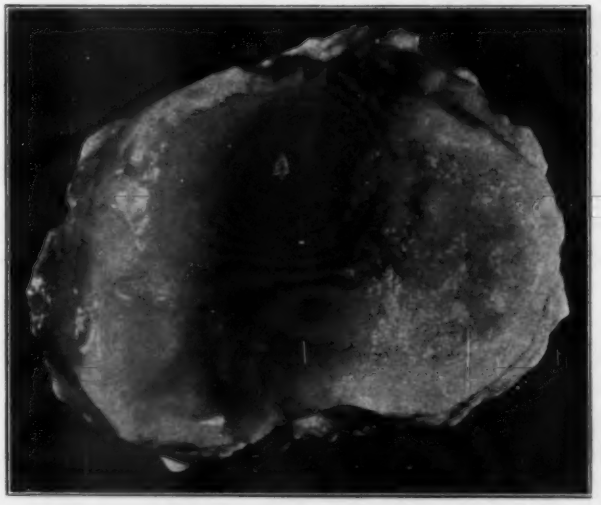

Fig. 4.—Advanced presenescent chondrosis. In the original color photograph it could be seen that normal opalescence was replaced by yellow areas at the sides and purplish areas in the center. Grossly there was no normal surface texture. Superficial and deep loss of substance, with fissures and clefts down to the underlying bone, may be noted.

arthrosis in the whole skeletal system. While such conditions are commonly diagnosed as arthritis, from the angles of approach and treatment this would be a clinical misconception.

In this lesion on gross inspection, the cartilage has a yellowish hue over its entire area, which also will show replacement of more or less of its glassily smooth glistening surface by a finely or coarsely granular one, with smaller areas of fissuring and fragmentation, and a ragged defect in full depth. Microscopically, the picture is one of generalized fibrillar degeneration and necrosis on which laceration or breakdown is superimposed.

These three pathologic types deserve individual consideration in prognosis and especially in treatment.

MECHANISM OF TRAUMA

1. Blow on patella in semiflexed weight bearing
2. Fall on flexed knee
3. Acute or recurrent dislocation patella
4. Residuals of patella fracture

After the primary pathologic condition is established, from any cause, ensuing weight-bearing function in flexion is continuously traumatic.

Since purely traumatic lesions have a high incidence, attention to the mechanisms of their production may be worth while. We believe in the logic of a therapeutic position which we have for some time maintained, that is, any reconstructive procedure for recurrent dislocation of the patella should include an arthrotomy and inspection of this structure, and except in children the best solution eventually of this disability in most cases is patellectomy.

Along this line observations of late complications of patella fractures, even clean transverse ones with apparent anatomic reduction and union, point to more consideration of the excellent functional results obtained by treating this injury primarily, as well as secondarily, by patellectomy.

SYMPTOMS

1. Sensation of squeaking or crackling; "catching"
2. Insecurity or weakness
3. "Giving way" with unaccountable falling
4. Stiffness and lameness on getting up on feet
5. Intermittent hydrops
6. Pain on kneeling or other pressure

The subjective symptoms of lesions of the patella cartilage are often so completely pathognomonic as to be alone diagnostic of the existence of the condition. It is true, however, that they are not always obtained without careful history taking, and it is true that some, but not all of them, obtain in other causes of internal derangement. Recognition by the patient of palpable or audible grinding on function,

plus pain on direct pressure on the patella, occurs only in the lesion discussed.

PHYSICAL SIGNS

1. Crepitation
   (*a*) Audible or palpable, subpatellar, on resisted quadriceps effort
   (*b*) To palpation on manual movement, with pressure, of relaxed patella in trochlear trough
2. Pain
   (*a*) On pressing or moving patella by hand in trough

Next, of course, in diagnosis come "physical signs," which as shown are few but absolutely diagnostic. To obtain the crepitation by the manual test, it is necessary to have the limb in supported full extension at the knee and to get complete quadriceps relaxation, which I find most easily obtained by rolling the limb in and out for a moment. While not specific, of course, an increase in joint fluid may prevail with this lesion. The physical examination should, of course, proceed step by step to complete anatomic and functional testing, and the diagnosis gains some support through exclusion of other lesions through both physical and roentgen examinations. Incidentally, this lesion is not demonstrable by roentgenogram, and logically so. It also can well be mentioned that more than one form of internal derangement with functional effects may prevail in any knee, and too frequently are arthrotomies carried out on the basis of one confirmed diagnosis and another lesion is left unsuspected to compromise the result.

COMPLICATIONS

The complications and sequelae of lesions of the patellar cartilage warrant consideration as having some bearing on treatment of election.

Quadriceps atrophy and weakness, especially after effusions
Chronic sympathetic synovitis
Joint laxity and secondary generalized degenerative arthrosis
Loose bodies from detached fragments of patellar cartilage
Traumatic secondary attritional erosion of condylar articular surfaces

Quadriceps atrophy and weakness are inescapable articular neuromuscular reflex responses. Since the knee joint is intrinsically weak and unstable and dependent to a major degree on this external muscular support, persistent and progressive quadriceps weakness leads to laxity and consequent joint deterioration, as well as to susceptibility to other traumatic derangement lesions.

It must also be realized that when the entire body weight, as in stair climbing or running, is taken on by a knee maintained at semiflexion by the resistance of the quadriceps, the pressure at the articular surface of the patella is extremely great. Under such conditions the sliding of a deeply abraded patellar cartilage surface can easily bring about

scoring or abrasion of the contacting articular cartilage of the femoral condyle, and at the very point where, when the knee is extended, it should smoothly glide on the anterior horn of the meniscus. Loosening or detachment of the latter by this mechanism has been found as a secondary complication.

TREATMENT

A. Palliative
   (1) Restriction of activity (stair climbing, running, kneeling)
   (2) Complete nonfunctioning joint rest in phases of generalized joint reaction
   (3) Medication or physical therapy entirely without effect, hyaline cartilage being devoid of any reparative powers

B. Surgical
   (1) Procedure according to type and age
      (*a*) Chondromalacia
         (1) Total scalpel shaving of surface down to 1/16 inch (0.16 Gm.) from bone (in second and third decades)
      (*b*) Traumatic chondritis
         (1) Shave of involved facet (in third and fourth decades)
         (2) Patellectomy (in fourth and fifth decades) or after fracture
      (*c*) Presenescent chondrosis
         (1) Patellectomy; full joint function assurable after patellectomy, but loss exposes condylar articular surfaces to traumatic hazards of earlier decades

Nonsurgical therapy at best can be only palliative and confined in influence to the subjective effects and to the extent of amelioration of them. If any reason on other counts prevails for deferment of surgical treatment, the proper regimen with recognition of the lesion at an early stage will permit postponement of operations without much risk for months or years.

However, with well advanced lesions, whatever the type and cause, surgical intervention alone can eliminate disability, restore full function and obviate unfortunate and irreversible sequelae.

As to operative methods, the prevailing pathologic condition indicates considerable discrimination. No confidence can be maintained as to the functionally efficient survival of any part of the cartilaginous structure of the patella in well established presenescent chondrosis. In middle life or beyond the relatively susceptible surface of the femoral condyles should be protected against the friction even of a well smoothed down patella. Since function is unimpaired by the loss of this bone, patellectomy is the only type of intervention to be considered. As to technic, the important point in the operation is synovial reapproximation, either vertically or from side to side, before the aponeurotic and capsular approximation is done.

In the after-care of this procedure, early motion is, of course, not feasible, and movement is begun gradually after the first ten days. At the end of another ten days the range of flexion will usually have progressed to not much more than 45 degrees and becomes arrested there. At this stage, with the patient under a brief relaxing anesthetic, motion to 90 degrees with release of fine adhesions under the suture line will occur through only the most gentle manipulation, and then on active use with progressive functional improvement can be counted on. At this stage resisted quadriceps reeducation, the *sine qua non* of the convalescent management of any knee arthrotomy, must be conscientiously adhered to.

As to operative indications, it might be held that the same logic applies to congenital chondromalacia. But there is this difference: In the second and third decades there is considerable reparative capacity in the most subchondral layer of this tissue, which has a good blood supply and capacity for fibrocartilaginous proliferation from the underlying bone. Secondly, at this phase of life and activities, the buffering protection of the patella against traumatic insults to the femoral condyles (which seems to be the only useful function of this sesamoid bone) would seem desirable to retain. As to technic, however, the nature of the basic pathologic condition clearly indicates, in conservative treatment of the lesion, a careful and meticulous scapel shaving of the entire patellar surface down to the basal cell layer. A localized curettage of the major defect will fail to relieve the existing symptoms as well as to forestall the inevitable progressive breakdown of the less grossly involved areas. While the result is not an absolutely quiet knee, it is symptomatically greatly improved and will continue so—at least it has done so in my experience for many years.

Finally, when traumatic lesions are residual conditions of patellar fractures, or complications of recurrent dislocation in adults, excision of the patella again is the choice to be made. Otherwise, in the great majority of cases, we have found a lesser extent of intervention to be satisfactory and, we believe, more advisable.

In the first place, such injuries are prone to occur in the physically and recreationally active phases of life, with the applicability of the argument for retention of this bone. In the second place, the lesion is a single and circumscribed one, confined to one facet of the articular surface, almost always the mesial one, and except when of very long standing it will not have penetrated to the full depth of the articular cartilage. Here again we want to oppose the idea or practice of simply curetting the ragged defect. The mechanics of functional movement of this articulating surface is such that it must be smooth and level or it will have the action of a tire tread against sliding on the pavement, and the defect will persist in its symptoms and will by marginal attrition

extend at its margins. The logical treatment is a uniform shaving down of the entire involved facet to the depth of deepest penetration of the traumatic defect.

SUMMARY

Lesions of the cartilage of the patella have a high incidence among the mechanical causes of disturbed or disabled function of the knee joint. Allowed to persist, they produce secondary progressive deteriorative and irreversible effects. The lesions are separable into three pathologic and etiologic types, and the differential discriminative surgical treatment of each is, with intelligent technic and after-care, highly successful in its results.

ARTERIAL BLOOD SUPPLY OF THE COMMON BILE DUCT

THOMAS C. DOUGLASS, M.D.
AND
WILLIAM W. CUTTER, M.D.
CHICAGO

WHILE studying the problem of the pathogenesis of stricture of the common bile duct we noted the lack of knowledge of its arterial blood supply. None of the standard anatomy texts describe the blood supply, and the illustrations in the various atlases are either very diagrammatic or lacking. The only reference in the literature so far discovered is in the report of postmortem dissections of Shapiro and Robillard,[1] who in the course of a study of the blood vasculature of the doudenum in 62 cases summarized the arterial supply from an undisclosed number of specimens. They found that the duct had a very poor supply of blood, consisting chiefly of end arteries and only an occasional anastomotic marginal artery. Injury to these small vessels, they said, might result in ischemic necrosis and stricture of the duct. This idea seemed to us to warrant further investigation.

MATERIAL AND METHODS

Fifty human postmortem specimens were taken from bodies during the course of the postmortem examinations. The excised specimens consisted of the gallbladder with a small segment of liver attached, the common duct and origins of the hepatic ducts, the entire hepatic trinity in the edge of the lesser omental bursa, the opened duodenum, the head and body of the pancreas, and the origins of the celiac and superior mesenteric arteries from the aorta. The specimens were then perfused with water to remove all the hemoglobin and bile possible. This was a time-consuming and often incomplete process, as the removal of all hemoglobin from the liver segment attached to the bile tract was almost impossible.

After perfusion, the arterial trunks were injected with a red synthetic latex, which produced a firm mass filling the arteries well. This material solidified after the specimen was fixed in 10 per cent formaldehyde solution so that the arteries might be sectioned without losing any of the injection mass. The specimen was washed to remove the formaldehyde. After dehydration in graded alcohols the specimens were cleared in a mixture of trio-o-cresyl and tributyl phosphate, so as to change the refractive index of the tissues according to a modifi-

From the Department of Surgery, Northwestern University Medical School.

Read at the Fifth Annual Meeting of the Central Surgical Association, Chicago, Feb. 20, 1948.

1. Shapiro, A. L., and Robillard, G. C.: Morphology and Variations of the Duodenal Vasculature, Arch. Surg. **52**:571 (May) 1946.

cation [2] of the method originally described by Spalteholtz.[3] In a few days most of the specimens were remarkably clear, so that tiny vessels might be seen clearly in the specimen. Attempts to use methyl salicylate for the clearing fluid failed because of persistence of hemoglobin in the liver and our inability to remove it, the combination of methyl salicylate and hemoglobin producing a black stain probably due to the conversion of hematin. A similar reaction between bile and methyl salicylate was noted.

Some difficulty was experienced in obtaining complete injection of the finer vessels because of the open ends of the vessels in the liver segment as well as the pancreatic section and the opened duodenum. This difficulty was overcome by carefully ligating the vessels opened, or by block removal of the intact liver, biliary tract, duodenum, pancreas and stomach. Even in those specimens not completely injected, the clearing made it possible to dissect the overlying tissues and expose and trace even the finest of arteries and arterioles from their source to the duct wall. These dissections were carried out with great care and the specimens numbered and sketched. The various patterns of blood supply were then classified and the accompanying illustrations are shown to demonstrate the observations.

OBSERVATIONS

For the sake of clarity the position of the organs in the illustrations are shown in an abnormal relationship. The duodenum has been rotated medially, and traction is applied in a caudad direction so as to show the common bile duct as entering the superior aspect of the duodenum. This is similar to the Kocher position or the embryologic position of these structures. The frequency of variations in the blood supply make any attempt to avoid vessels by a particular surgical approach to the duct unreliable. These drawings are also semidiagrammatic as concerns the bile tract, since no attempt was made to study the variations so well known in these structures.

All blood vessels are drawn to a rough scale. Four distinct size classifications were made. The smallest vessel described is represented by a single thin line on the drawings and indicated a very small artery, an arteriole or an arterial capillary. The vessels supplying the duct were of this caliber. The second vessel in increasing size is represented by the cystic vessels, the posterior pancreaticoduodenal and the right gastric artery. Slightly larger but in the same general classification were the right gastroepiploic and sometimes the right or left gastric arteries. In the third order are the gastroduodenal and the terminal branch of the right or left hepatic artery. Slightly larger but in the same general classification are the common hepatic and the splenic arteries. The largest, or fourth order, includes the hepatic, the superior mesenteric and the slightly larger celiac.

---

2. Groat, R. A.: Clearing Tissues with Mixtures of Tributyl and Tri-o-Cresyl Phosphates, Stain Technol. **16**:111, 1941.

3. Spalteholtz, W.: Ueber das Durchsichtigmachen von menschlichen und tierischen Praparaten und seince theoretischen Bedingungen, Leipzig, S. Hirzel, 1911.

In the great majority of specimens (42 cases, or 84 per cent) an anastomotic loop or loops were formed similar to that seen in the arterial supply of the intestinal tract. Since the common bile duct is a similar hollow viscus, the parallel is readily seen. Variations were noted so frequently in all the vessels in this region that aside from the loops mentioned, which also varied in origin, no regular pattern was discernible. In the remaining 8 specimens there was no anastomotic loop, but the blood supply could be said to be adequate for a structure the size of the common bile duct. Figures 1, 2 and 3 illustrate variations

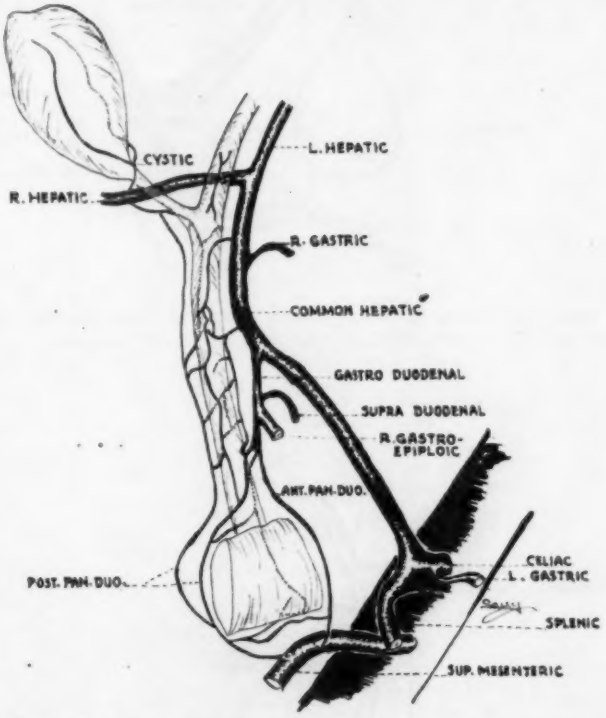

Fig. 1.—Extrahepatic biliary tract with arterial supply, with the duodenum rotated medially. Typical arterial supply was found in 84 per cent of cases with marginal anastomotic loop. This illustrates supply by posterior superior pancreaticoduodenal (100 per cent), right hepatic (84 per cent), posterior inferior pancreaticoduodenal (56 per cent) and common hepatic (40 per cent) arteries.

in what is regarded as the most frequently occurring pattern. In these specimens a richly anastomosing network is seen covering much of the surface of the duct. Figure 4 is an example of the 16 per cent of specimens showing no distinct anastomotic loop. It is seen that even in this specimen the blood supply is adequate and that nothing short of skeletonization of the duct would deprive any material segment of its blood supply.

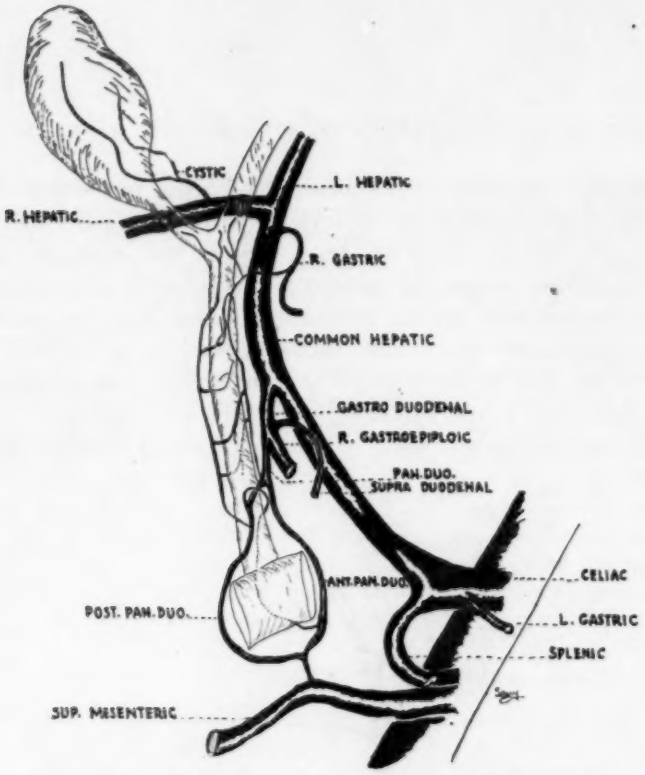

Fig. 2.—Marginal loops. This illustrates supply by right gastric (44 per cent) and anterior inferior pancreaticoduodenal (32 per cent) arteries.

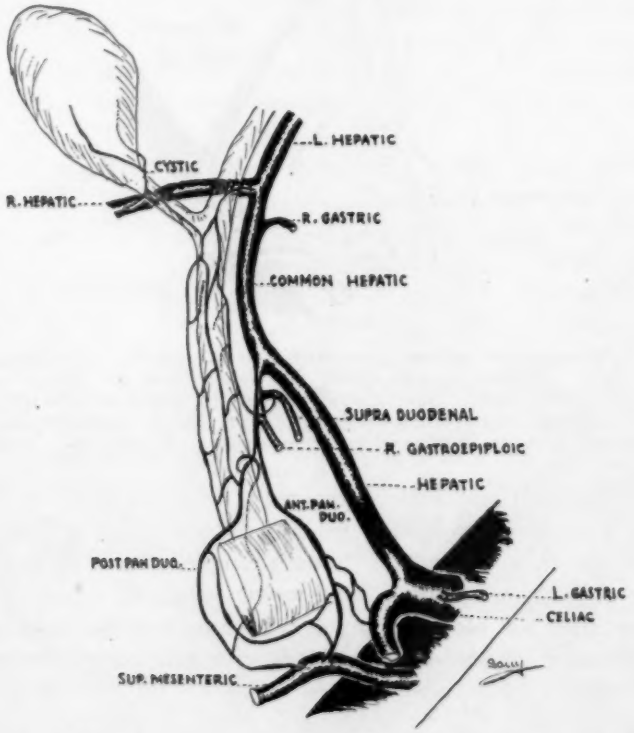

Fig. 3.—Another pattern showing marginal anastomotic loops, showing also supply by cystic (28 per cent), supraduodenal (24 per cent) and left hepatic (6 per cent) arteries. Anastomosis between splenic and pancreaticoduodenal systems (90 per cent of cases) may be noted.

The anastomotic loops formed on the right side of the duct, covering the upper two thirds, consisted of the right hepatic and the posterior superior pancreaticoduodenal and/or the posterior inferior pancreaticoduodenal. In specimens when this loop was on the left side of the upper two thirds of the duct the common hepatic, the right hepatic and/or the posterior superior pancreaticoduodenal contributed branches. An anastomotic loop was also found covering the lower third of the duct on both sides formed by the posterior superior pancreaticoduodenal

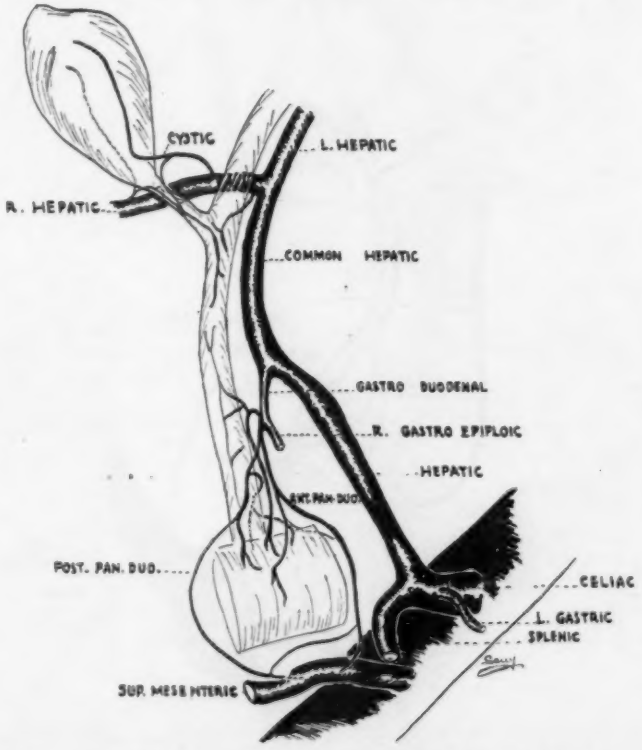

Fig. 4.—No anastomotic loops (16 per cent of cases), showing separate origin of anterior and posterior cystic arteries (6 per cent of cases).

and/or the anterior superior pancreaticoduodenal with either the posterior inferior pancreaticoduodenal or, more commonly, the anterior inferior pancreaticoduodenal.

A brief statistical summary of those vessels supplying the bile duct with branches is given in the following paragraphs, with references to the appropriate illustrations.

Posterior Superior Pancreaticoduodenal Artery.—The posterior superior pancreaticoduodenal artery supplied branches to the duct in all cases studied (all figures).

In 49 instances the posterior superior pancreaticoduodenal artery crossed the duct anteriorly and in 50 cases gave some blood supply to the duct. In 1 specimen the vessel passed the duct posteriorly and anastomosed with an anomalous right hepatic artery (fig. 5).

Right Hepatic Artery.—The right hepatic artery supplied branches to the duct in 84 per cent of the specimens. In 38, this vessel had originated from the common hepatic artery (fig. 1); in 4, from the

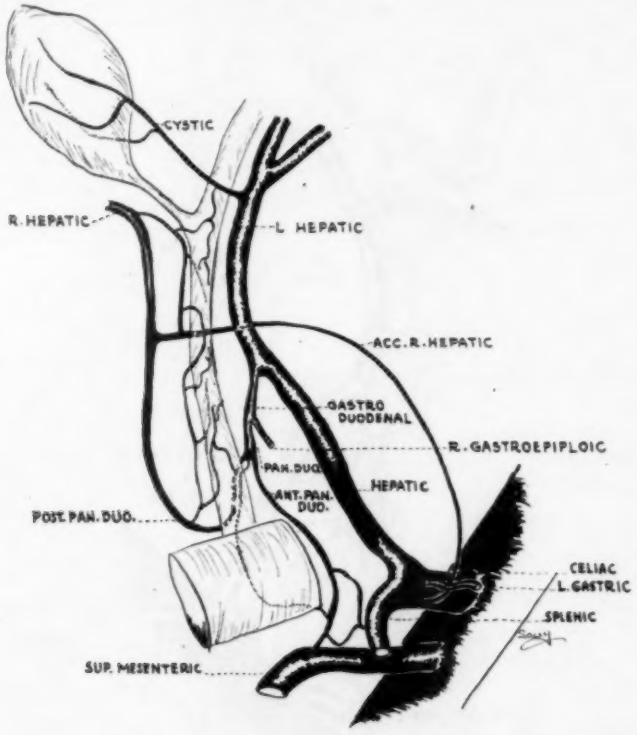

Fig. 5.—Anomalous right hepatic artery arising from posterior pancreaticoduodenal. A branch from the celiac artery supplies the duct. The posterior superior pancreaticoduodenal artery passes posterior to the duct (2 per cent of cases). Splenic anastomoses may be noted.

superior mesenteric (fig. 6), and in 1 instance as the first branch of the celiac (fig. 5).

Posterior Inferior Pancreaticoduodenal Artery.—The posterior inferior pancreaticoduodenal artery gave branches to the duct in 56 per cent of the specimens studied. In 25 instances this vessel had a normal origin from the gastroduodenal artery (figs. 1 and 2), in 2 it arose as a separate vessel from the superior mesenteric artery (figs. 7 and 8), and in one it was the first branch of the celiac (fig. 9).

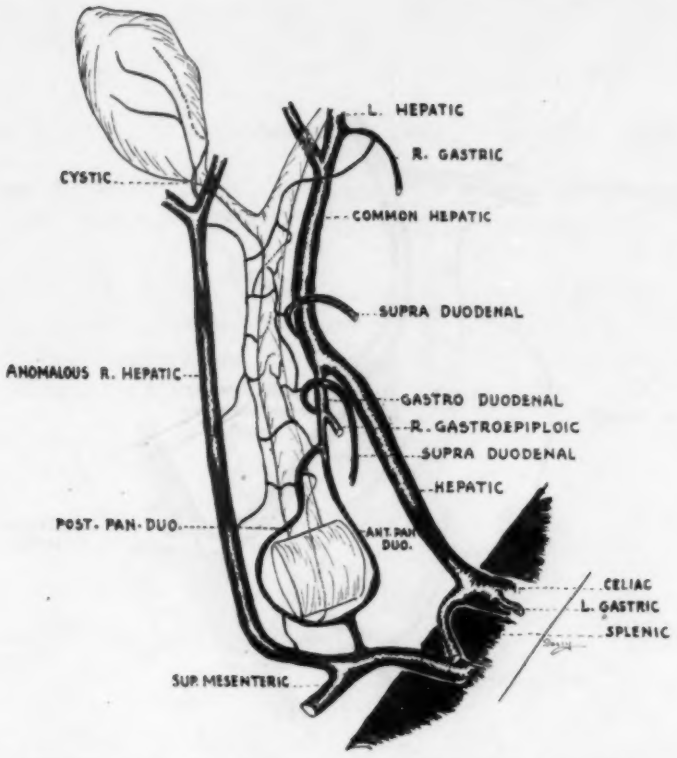

Fig. 6.—Anomalous right hepatic artery arising from superior mesenteric. Two supraduodenal arteries, both supplying the duct, may be noted. The right gastric artery also supplies the duct.

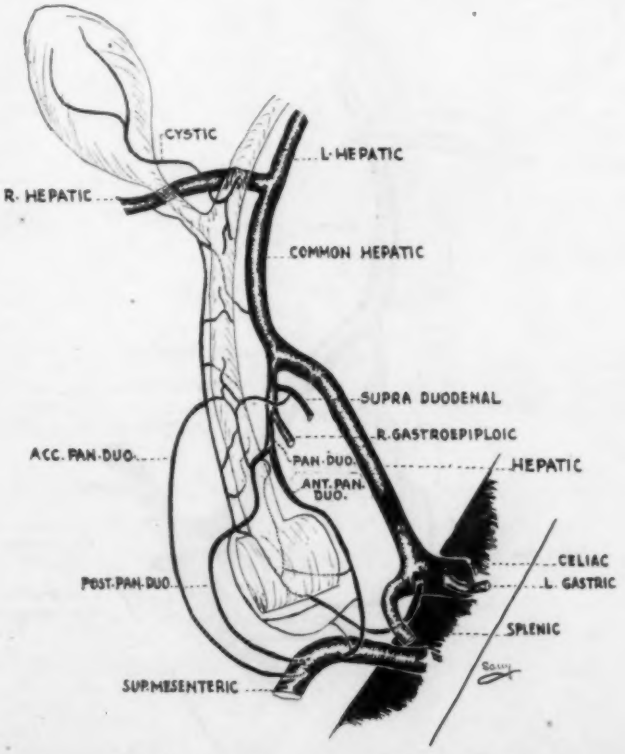

Fig. 7.—Accessory posterior inferior pancreaticoduodenal artery supplying the duct. Anomalous pancreaticoduodenal artery arising from the celiac artery may be noted.

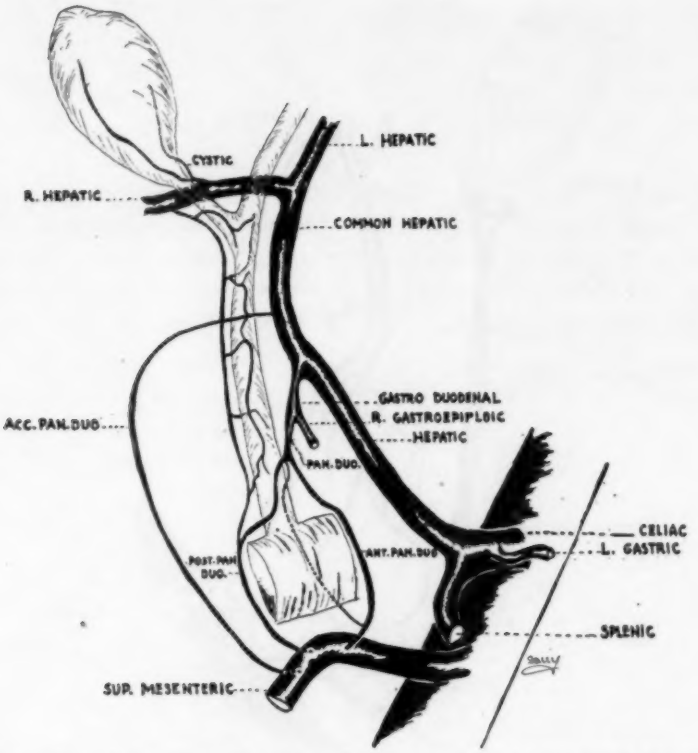

Fig. 8.—Superior mesenteric artery supplies the duct (4 per cent) by an accessory pancreaticoduodenal artery.

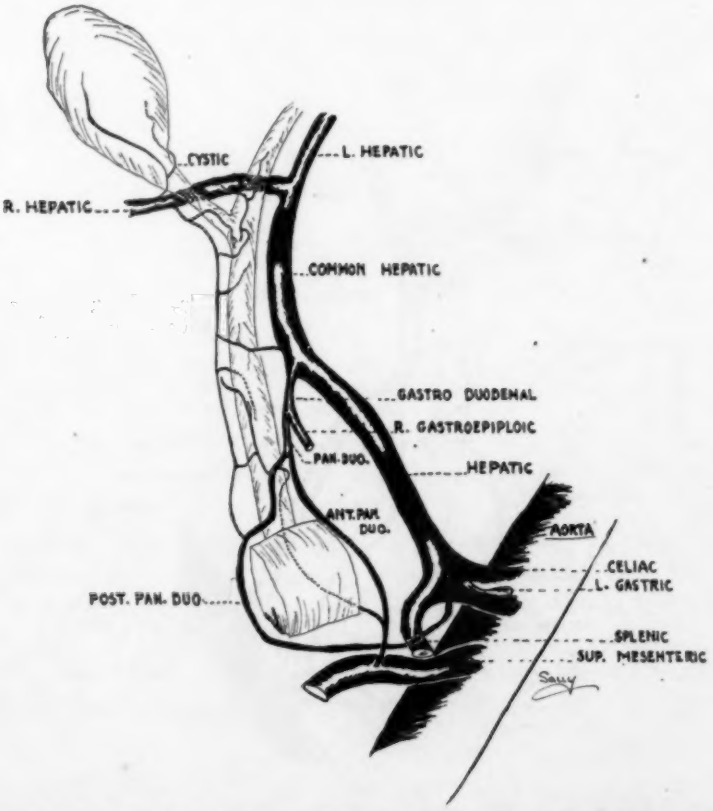

Fig. 9.—Posterior inferior pancreaticoduodenal artery arising from the celiac artery. This illustrates the need for care in clamping the cystic duct to avoid injury to arteries of the duct.

Common Hepatic Artery.—The common hepatic artery gave branches to the duct in 20 instances, or 40 per cent of the specimens studied (fig. 1).

Right Gastric Artery.—The right gastric artery gave some supply to the duct in 22 instances, or 44 per cent of the specimens studied. Of these the gastric artery was normal in origin, that is from the common hepatic distal to the origin of the gastroduodenal artery, in 18 (fig. 2),

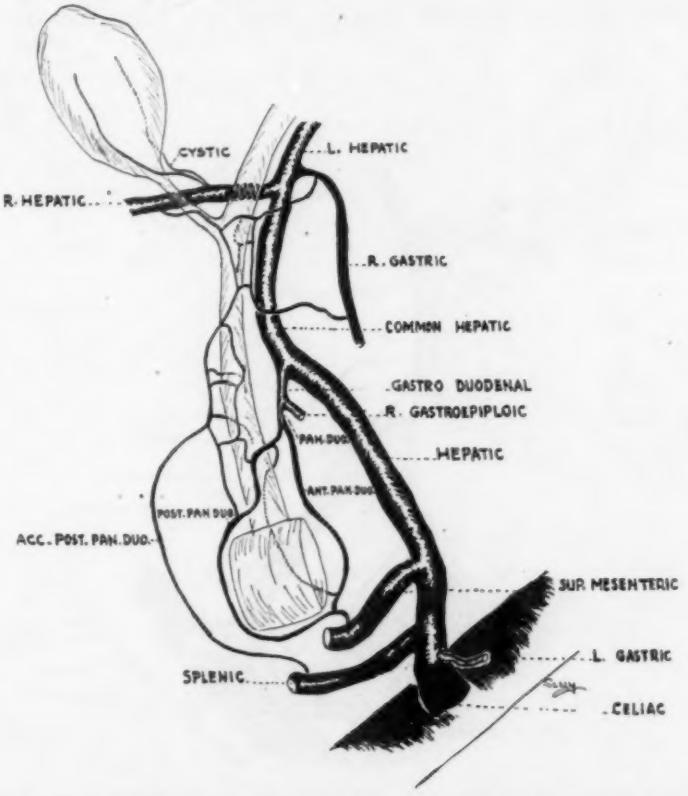

Fig. 10.—Splenic artery supplying the duct by accessory posterior inferior pancreaticoduodenal artery. The anastomotic loop of the right gastric artery giving three branches to the duct may be noted. The superior mesenteric artery here is a branch of the celiac artery.

arose from the left hepatic artery in 2 (figs. 6 and 10), arose in conjunction with the supraduodenal artery in 1 and gave a branch to the duct before bifurcation (fig. 11), and in 1 instance formed a loop with itself parallel with the common duct (fig. 10).

Anterior Inferior Pancreaticoduodenal Artery.—The anterior inferior pancreaticoduodenal artery supplied branches to the duct in 16

instances, 32 per cent, and in every instance to the posterior surface of the duct (fig. 2), and in 1 specimen it supplied a branch anteriorly as well.

Cystic Artery.—The cystic artery supplied branches to the duct in 14 specimens, or 28 per cent. In 10 of these the branch to the common bile duct was given off before bifurcation into the anterior and posterior branches to the gallbladder (figs. 3 and 5). In 3 instances the origin of the cystic artery was bifid, the anterior and posterior branches to the gallbladder having separate origins from the right hepatic artery

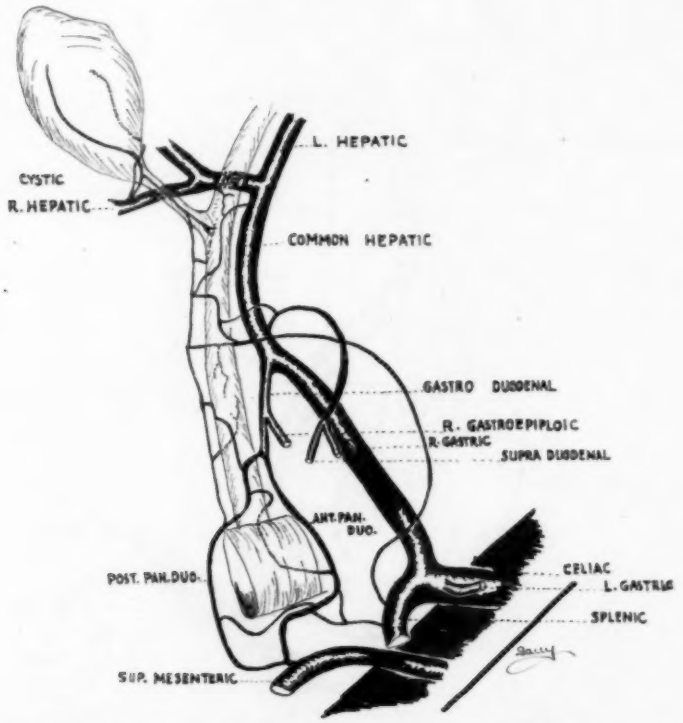

Fig. 11.—The right gastric and supraduodenal arteries have a common origin which supplies the duct before bifurcation. Supply of the duct by the splenic artery may be noted. This illustrates the necessity for separate and distal ligation of the cystic artery during cholecystectomy.

(fig. 10). In 1 of this latter group of specimens the anterior branch alone supplied a branch to the duct (fig. 12). In 1 the branch to the common bile duct was given off from the anterior branch of the cystic artery after its bifurcation (fig. 11).

Supraduodenal Artery.—The supraduodenal artery gave branches to the common bile duct in 12 instances, 24 per cent. In 10 of these the supraduodenal artery had a normal origin from the gastroduodenal

artery. In 2 instances there were two supraduodenal arteries, both giving branches to the duct, in one arising from the posterior superior pancreaticoduodenal and the anterior superior pancreaticoduodenal, respectively (fig. 4), and in the other from the common hepatic and gastroduodenal.

Anterior Superior Pancreaticoduodenal Artery.—The anterior superior pancreaticoduodenal artery gave branches to supply the duct in 12 instances, 24 per cent, all to the posterior surface (fig. 12) while in 4, branches were present anteriorly as well.

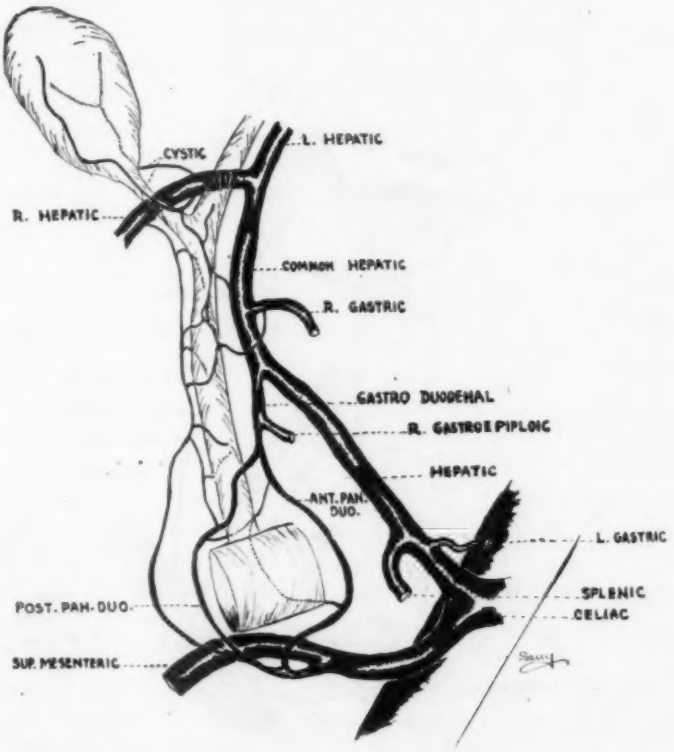

Fig. 12.—The duct is supplied by the anterior superior pancreaticoduodenal artery (24 per cent of cases). The common ostium of the celiac and superior mesenteric arteries may be seen. This also illustrates the advisability of the distal ligation of the cystic artery to avoid injury to the branches to the duct.

Left Hepatic Artery.—The left hepatic artery supplied branches to the duct in 3 instances, 6 per cent.

Gastroduodenal Artery.—The gastroduodenal artery was found to supply the duct in 1 specimen, 2 per cent.

Splenic Artery.—The splenic artery supplied the duct in 2 instances, or 4 per cent. In 1 it supplied the duct directly after following the course of the proper hepatic and then anastomosed with the loop on the right

side of the duct (fig. 11) and in the other with a direct anastomosis with the loop on the right side of the duct (fig. 10). It was also noted that in 90 to 94 per cent of specimens some anastomosis was present between the splenic and the pancreaticoduodenal arterial system, while in 22 per cent of specimens this anastomosis was direct and by means of a large caliber vessel.

Celiac Artery.—The celiac artery supplied a branch to the common duct in 1 specimen which was by means of a large second order

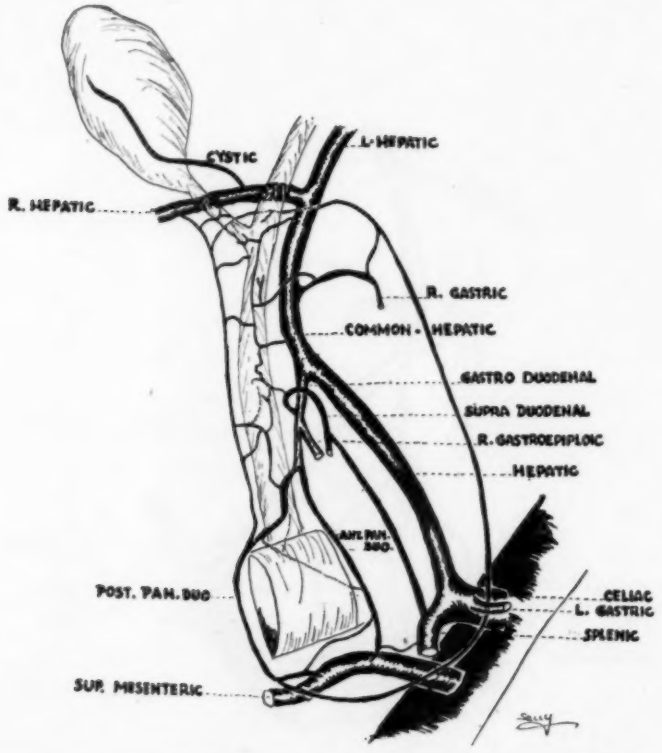

Fig. 13.—Anomalous branch of posterior inferior pancreaticoduodenal artery supplying the duct. Large caliber anastomosis between splenic and supraduodenal arteries may be noted.

vessel arising as an anomalous first branch of the celiac, proceeding directly posterior to the duodenum through the substance of the pancreas and supplying the common duct posteriorly in its distal portion.

Superior Mesenteric Artery.—The superior mesenteric artery supplied the duct in 2 instances, 4 per cent, by means of an anomalous second order vessel arising in conjunction with the posterior inferior pancreaticoduodenal artery (fig. 13).

COMMENT

In none of our specimens was the hepatic artery proper, the left gastric artery, or the inferior phrenic arteries involved in the supply of the common bile duct.

It is of interest to note that in 3 instances in our series the celiac and the superior mesenteric vessels had a common ostium.

From these figures it seems clear that the blood supply of the common bile duct is generous and follows a pattern similar to but less regular than that seen in the intestinal blood supply. As might have been predicted those vessels closest to the structure supplied the duct in the highest percentage of specimens and those more remote less frequently. These observations are at variance with those of Shapiro and Robillard, who found an anastomotic loop only occasionally as opposed to 84 per cent in our series. The short vessels which they interpreted as end arteries were not frequent in our specimens; the vessels generally pursued a course parallel with the duct giving off branches along their course.

The irregularity of the blood supply might have been expected from the variation reported in this region by Anson and his co-workers.[4]

SURGICAL APPLICATIONS

Since the blood supply varies so widely in the 50 specimens studied and the study of some 500 or more specimens would probably disclose other undescribed variations, it seems obvious that no single surgical approach to the duct could be accomplished safely without careful dissection to avoid these small vessels. Gentle surgical technic with adequate light, good anesthesia and painstaking dissection in this region should enable the competent surgeon to avoid dangerous injury to the vessels supplying the duct. Further, it would seem desirable to strip as little of the peritoneum from the duct as is compatible with satisfactory and safe exposure during cholecystectomy or choledochostomy to avoid injury to these vessels.

While serious injury to the duct during operation, such as crushing with a hemostat or partial ligation of a segment of the wall, may cause necrosis of the duct wall and subsequent leakage and scar tissue replacement with resultant stricture, it seems unlikely that in the absence of these injuries the blood supply would be so seriously damaged as to cause an ischemic necrosis as suggested by Shapiro and Robillard. Certainly due care in the exposure of the duct should circumvent this possibility.

4. Daseler, E. H.; Anson, B. J.; Hambley, W. C., and Reimann, A. F.: The Cystic Artery and Constituents of the Hepatic Pedicle, Surg., Gynec. & Obst. **85**:47, 1947.

Further support is given to this belief when one notes that properly performed choledochostomy or choledochotomy is rarely if ever followed by stricture of the common bile duct, which would hardly be expected to be true if minimal injury could produce an aseptic necrosis.

When a segment of the duct is inadvertently removed during the course of a cholecystectomy the continuity must be reestablished. Recently Cattell [5] reemphasized the desirability of end to end anastomosis when this is possible without tension. In following this procedure only the barest minimum of duct ends should be freed from the surrounding fat and peritoneal covering in order to minimize the damage to the blood supply. The stripping of long segments of the duct of their vessel-bearing tissue could very well account for some of the failures reported in these cases, since an aseptic necrosis of the suture line and fibrous structure might be expected to follow such a procedure.

The current practice of separate ligation of the cystic artery during cholecystectomy is obviously correct, and this should be carried out as far distal as practical in order to save branches to the duct in this region (28 per cent in our series).

SUMMARY AND CONCLUSIONS

The arterial blood supply of the common bile duct has been dissected and described in 50 fresh postmortem cleared specimens. An abundant anastomotic loop was present in 84 per cent of the specimens studied and was similar to the arrangement of the blood supply of the bowel or ureter. No anastomotic loop was found in 16 per cent of the specimens.

Of the vessels supplying the duct the most frequent are the posterior superior pancreaticoduodenal (100 per cent), the right hepatic artery (84 per cent), the posterior inferior pancreaticoduodenal (56 per cent), the right gastric (44 per cent), the common hepatic (40 per cent), the anterior inferior pancreaticoduodenal (32 per cent), the cystic (28 per cent), the supraduodenal (24 per cent) and the anterior superior pancreaticoduodenal (24 per cent). Pronounced variation in the origin of the vessels supplying the duct was the rule in these specimens. In no 2 instances was the blood supply identical.

Suggestions are offered to minimize injury to the blood supply during surgical treatment of the biliary tract.

5. Cattell, R. B.: Benign Strictures of the Biliary Ducts, J. A. M. A. **134**:235 (May 17) 1947.